PELVIC INFLAMMATION IN WOMEN

Uterus Removed from Patient Dying of Streptococcic Endometritis—Postabortal Type, Placenta in Situ.

Kidney from Patient Who Had Streptococcic Endometritis, Removed at Autopsy, Showing How the Kidney Participates in Virulent Infections.

PELVIC INFLAMMATION IN WOMEN

BY

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PREFACE

This monograph is presented to the medical profession in the hope that it may serve a purpose, i.e., to awaken in both the practitioner and the specialist a keener appreciation of the importance of that large group of diseases peculiar to women which result from septic gonorrhoeal and tuberculous infections.

Nowhere in the English or American literature is the subject presented in its entirety, and while the author does not presume to present a résumé of all the literature on the subject, he has attempted to describe concisely and consecutively the common inflammatory lesions affecting the genital organs in women.

While some of the statements may appear dogmatic and have been repeated several times in the text, many years of clinical teaching has convinced the author that there are certain positive truths in medicine which cannot be taught except by positiveness and repetition.

In this work the effort has been made to give the reader the accepted pathology, a definite clinical picture and a description of the treatment which has best served the author in his special practice.

I take this opportunity to express my appreciation and thanks to my associates, Drs. Harvey B. Matthews, Alfred C. Beck and George W. Phelan, who have aided me materially by their detailed study of case histories and their helpful criticism, and to my artists, Mr. Howard Shannon and Miss Emily Freret, as well as to my publishers, who have so courteously coöperated with me in making this monograph a reality.

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Infections of the female genital organs make up such a large group of the diseases peculiar to women, and their pathology and treatment are so distinct because of the special anatomy of the genital tract, that it is but proper that these infections should be discussed apart from the other pathological conditions which may affect the female genitalia.

Before taking up a discussion of the inflammatory diseases which are directly the result of labor, abortion, and mixed or gonorrheal infection spreading from acute cervical and endometrial lesions, we will review the infections of the vulva, introitus, vagina, and cervix, and thus give the reader a consecutive view of the acute and subacute inflammations as they involve the successive tissues. In acute gonorrheal infection as Skene's glands, and the ducts of Bartholin, are the most common points of infection.

INFECTIONS OF SKENE'S GLANDS

Skene's glands, or the para-urethral ducts, are two small tubules which lie on each side near the floor of the female urethra and extend up from the meatus urinarius for about three fourths of an inch; these ducts open just anterior to the center of the urethral lips. Koch maintains that they represent the lower extremities of the wolffian ducts; other authorities, however, maintain that they are simply exaggerated lacunae. Their chief interest lies in the fact that *gonorrheal* invasion of these ducts *is the most persistent lesion* with which the gynecologist has to deal, for the gonococcus may remain indefinitely buried beneath the lining

cells of the tubule and may cause a chain of symptoms referable to the urinary tract, as chronic urethritis, with its frequent and burning urination, or a persistent infective discharge, which in turn carries the infection higher up to the cervix and uterus.

Diagnosis.—The diagnosis is made by exposing the mouth of the tubules. This is done by everting the urethral mucosa, the finger in the vagina making pressure against the urethra from within outward along its course. By this manipulation a drop of pus may be expressed from the mouth of each tubule, which is situated just within the meatus on either side of the floor of the urethra. *The presence or absence of the gonococcus should always be demonstrated* in the pus *by the microscope* before treatment is instituted. Occasionally the outer orifices of the ducts may be closed as a result of the inflammatory process, when an abscess may form in the tubule and suburethral tissues. The abscess usually discharges spontaneously *through the tubule.*

Fig. 1.—Everting the Mucosa of the Urethra with the Finger in the Vagina and Milking Skene's Ducts of Pus.

Treatment.—Destruction of gonococcic infection in this location may be accomplished either by (a) injection or (b) cauterization. After cocainizing the meatus and the urethra, by inserting into the urethra a pledget of cotton wet with a ten per cent solution of cocain, which is left in place for five or ten minutes, a probe pointed hypodermic needle is inserted into the duct, and a drop of pure carbolic acid, or 25 per

cent silver nitrate solution, is injected into the tubule. The treatment may have to be repeated several times at intervals of about a week. A more satisfactory method is that of splitting open the ducts with the electric cautery. Under local anesthesia a fine pointed cautery knife is passed into the duct, the current turned on, and the tubule slit upward into the urethral floor. The tract is then cauterized, destroying the tubule and its contents.

INFLAMMATION AND ABSCESS OF BARTHOLIN'S DUCTS AND GLAND

The Bartholin duct, which is found just within the vulvovaginal orifice, opens external to the hymen in nulliparous, and below the caruncle in parous women. It is the external outlet of the gland; the vulvovaginal gland lies, as a rule, behind the anterior layer of the triangular ligament, or may lie behind or in front of the posterior layer. In structure it is a compound tubular gland, which secretes a thin translucent fluid and is the homologue of Cowper's gland in the male. Both glands and ducts are lined with tall columnar epithelium. The duct alone may be involved, or the infection may extend to the gland. It is usually a bilateral involvement. When the inflammation is confined to the duct and its lining epithelium, the orifice of the duct is red and swollen and a purulent discharge escapes on pressure. Long after all of the acute manifestations of the infection have disappeared, *the red raised macula about the orifice* will point to the origin of the infection, for it seems *impossible without destruction of the duct to completely eradicate the latent gonococcus*. The infection extends along the duct to the gland, and, as a result of the inflammation, the tube lumen may become obliterated, and a collection of pus forms within the gland itself, distending it and forming an abscess. The bacteriology of the abscess content will usually show staphylococci alone or in combination with the gonococcus.

Symptoms and Physical Signs of Bartholin Gland Infection. —The patient complains of a vulvar swelling on one or both sides of the labia minora, and other general symptoms of a local inflammation: on inspection, the usual evidences of inflammation, such as redness and swelling, are to be seen. The orifices of the duct or ducts are red and swollen, and there is an ovoid tumor in one or both labia, more or less occluding the orifice of the vagina. The overlying tissues of the labia are red, hot, and painful, and the tumor rapidly enlarges; the enlargement extends backward toward the commissure and upward into the submucous tissues of the vagina. There is usually a slight elevation of temperature.

Abscess of the Bartholin gland must be differentiated from a pudendal

hernia, a retention cyst of the vulvovaginal gland, inclusion cysts of the lower vaginal wall, and vulvar tumors. A hernia presents none of the pain and sensitiveness of a gland infection, neither is there the history or other evidence of gonorrheal exposure; and usually one or more of the usual signs of hernia are demonstrable in the tumor, namely, an impulse on coughing, reducibility in the recumbent position, evidence of intestinal obstruction, and resonance on percussion. A retention cyst of the vulvovaginal gland has none of the inflammatory symptoms which characterize an abscess, the swelling is ovoid but not painful, its growth is slower and the overlying skin and mucosa may be moved freely over it. Aspiration of the contained fluid will always make the diagnosis positive. Inclusion cysts of the lower vaginal wall are rare, usually solitary, and vary in size from that of a pea to that of a cocoanut. The mucous membrane of the vagina moves freely over the surface of the tumor. If the cyst is on the posterior wall, rectovaginal touch will quickly reveal its limitations. Vulvar tumors are distinguished by their density and usually are devoid of inflammatory phenomena.

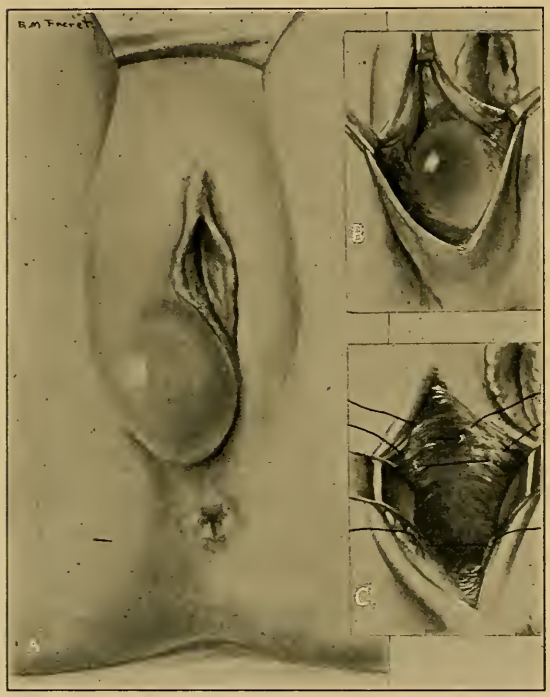

FIG. 2.—A. INFECTED CYST OF BARTHOLIN'S GLAND, EXTENDING BACKWARD TOWARD THE COMMISSURE. B. INCISION EXPOSING DISTENDED GLAND. C. GLAND REMOVED—SUTURES IN PLACE.

TREATMENT.—It may be possible to dilate the Bartholin duct with a probe and allow the contained purulent fluid to escape. A probe pointed hypodermic needle, attached to a syringe containing a dram of a 25 per cent argyrol solution, may then be passed through the duct and injected into the gland cavity. This treatment may be repeated daily, if the lumen of the duct will permit. Unfortunately it is painful and is neither as satisfactory nor as permanent as incision or excision of the abscess cavity.

Incision.—After thoroughly preparing the vulva by cutting away the hair, cleansing the region of operation, and then painting it with tincture of iodine three and one half per cent, the tissues about the tumor should be infiltrated with a one half per cent solution of novocain. When the anesthesia is complete, the abscess may be emptied by a free incision over the gland on its mucous surface, just within the labia, at the junction between the skin and mucous membrane. While this allows the escape of pus, extirpation or destruction of the gland membrane is necessary to prevent recurrence. This may be done with a sharp curet, or by the application of pure carbolic acid to the abscess cavity. After the capsule has been removed or destroyed in this way, a firm gauze pack should be left in situ for several days. In the author's hands nitrous oxide and oxygen anesthesia is preferable to local infiltration.

Excision of the Gland is applicable alike to abscess or cyst, and when done properly, has many advantages over incision, as it insures extirpation of the entire gland. Under local or general anesthesia, the abscess cavity or cyst may be emptied of its contents through an aspirating needle; then, without withdrawing the needle, the cavity is injected with melted paraffin wax; this promptly solidifies within the limits of the gland and makes it possible to remove the entire sac without perforating the gland capsule. After the paraffin has set, an incision parallel to the labia is made through the mucocutaneous margin and the gland enucleated by blunt dissection. After the control of bleeding by pressure, or by ligature of the individual vessels, the wound may be closed by sutures, providing drainage, by the insertion of a narrow strip of rubber tissue in the posterior angle of the wound.

INFLAMMATION OF THE VULVA

Vulvitis is an inflammation of the tissues making up the vulva. It may be studied under the following headings: 1. Simple vulvitis, 2. gonorrheal or specific vulvitis, 3. follicular vulvitis, 4. diabetic vulvitis.

Simple Vulvitis.—Simple vulvitis is an inflammation of the vulva which is characterized by free serous or mucopurulent discharge, swelling, and heat. It may be either acute or chronic.

Causes.—It may be caused by uncleanliness, traumatism, chafing, or parasites (as the pediculi pubis, seat worms, or saprogenic microbes), or by irritating discharges from the uterus, vagina, or bladder, or by excessive coitus, or masturbation, or diathesis. It is comparatively rare and is seen most often in children and young girls with delicate epithelial surfaces and in obese women.

Symptoms.—In the acute non-specific form the patient complains of

local irritation, tenderness, and pain and smarting on urination as the urine passes over the inflamed areas. The labia are painful, swollen, red and hot. The discharge is profuse and mucopurulent and has a fetid odor; and the external vulvar surfaces and inner surfaces of the thighs become excoriated, primarily from scratching due to the existing pruritus. A dermatitis is likely to follow. Occasionally the mucous glands are obstructed and a form of acne develops. The inguinal glands may be enlarged and cause pain in both groins.

In chronic vulvitis the itching and burning are prominent symptoms. The itching may be so severe as to preclude rest. There is less swelling, the discharge is thinner, of a serous character, and less profuse, but excoriations due to scratching are constant lesions; in fact the inner surfaces of the thighs are often inflamed and eroded.

Treatment.—The treatment of simple acute vulvitis may be summarized as follows: (1) Removal of the cause, (2) physical rest, (3) frequent cleansing and local medication. Irrigations with a warm borax solution, one dram to the quart, for cleansing. Care must be exercised that none of the solution enters the vagina and forces the infection higher up. Sitz baths of borax water of one half to one hour duration help to reduce the local swelling.

They should be taken twice daily and followed by rest in the recumbent posture. Sedative lotions, like lead and opium, or a saturated solution of aluminum acetate, applied to the inflamed parts, tend to relieve the pain. Separation of the labia with antiseptic compresses secures free exit for the secretions and prevents agglutination of the labial edges. A saline laxative should be taken each night before retiring to secure free action of the bowels; while the urine must be rendered bland and non-irritating by the copious ingestion of alkaline waters. Food likewise should be light and nourishing, without condiment. Coffee and all alcoholic beverages should be prohibited.

Chronic Vulvitis.—The same general rules, as to cleanliness and the condition of the urine, apply in the treatment of chronic cases. The cause, however, is more easily determined, hence the treatment is less empiric. When the vulvitis is due to parasites, oleate of mercury and mercurial ointment are useful.

Irritating discharges from the vagina, cervix, or uterus, should receive appropriate local treatment. Diatheses demand proper local and hygienic attention. Excessive coitus or masturbation should be prohibited. Local measures for the correction of the associated dermatitis and pruritus are usually required. These consist of thorough cleansing of the vulvar surfaces with "Synol" soap, irrigation and the application of mild astrin-

gents and antiseptics, as weak solutions of permanganate of potassium, mercuric bichlorid, carbolic acid. Lint compresses wet with an aqueous solution of argyrol should be placed between the labia to keep them separated. As the dermatitis subsides the parts should be kept dry and dusted with bland powders, as stearate of zinc, cornstarch, or lycopodium.

Gonorrheal Vulvitis.—This is a specific inflammation of the vulva caused by the gonococcus. It is estimated that about 75 per cent of the cases of vulvitis are of gonorrheal origin. It is characterized by an increased burning during micturition, profuse purulent discharge, and redness of the meatus and the orifices of Bartholin's ducts. The disease has a marked tendency to spread to the adjacent structures, involving the urethra, cervix, uterus, tubes and ovaries. The lesions and symptoms are usually of greater intensity than are found in the simple type. Gonorrheal vulvitis may be epidemic in children, the disease running through schools and hospital wards. The presence of the hymen seems to act as a protection to the little girl against upward invasion of the gonococcus, though the urethra, Bartholin's and Skene's glands are all promptly invaded.

Symptoms.—Within a few days after suspicious coitus or exposure to infection, the vulva becomes irritated and heat, pain, redness and swelling of the vulvar tissues are noted; the pathologic changes are always more pronounced than in non-specific vulvitis. The urethra rapidly becomes involved and the gonococcus may be determined by the microscope. Gonorrheal infection, except in children, generally extends or is carried by the treatment, to the internal genital organs. The inflammatory reaction is most severe in virgin tissues. The vulvovaginal glands (Bartholin's) and the Skene's tubules just within the meatus are almost always infected in a gonorrheal vulvitis. No case of acute vulvitis should be classed as gonorrheal, no matter how strong the clinical evidence, unless the gonococcus can be demonstrated in the discharge; and yet the absence of the gonococcus is not positive proof against the specific character of the disease, as the gonococcus is capable of assuming amorphous forms and remaining quiescent for years, only to resume its original form and virulence under proper irritation.

Treatment.—The treatment of acute gonorrheal vulvitis presents four definite purposes on the part of the attendant, (1) *to obtain local cleanliness,* (2) *to prevent extension of the infection to other structures,* (3) *to eradicate the infection by destruction of the gonococcus,* and finally (4) *to impress upon both patient and nurse the contagiousness of the disease and the dangers of accidental infection of eyes, rectum, etc.* Patients suffering from acute gonorrheal vulvitis should be placed in bed

in the Fowler position, as rest and postural drainage definitely minimize the danger of upward extension. In children and nulliparous women care must be exercised that no application or examination be carried past the hymen, for specific infection of the vagina and cervix spreads to the cervix and uterus with great virulence. Separation of the labia with the fingers of the gloved hand exposes the inflamed parts, and careful irrigation with solutions of normal saline or weak permanganate of potassium, will remove the purulent discharge. The irrigation should be repeated several times daily, after which the affected surfaces should be carefully dried and painted over with a two per cent solution of argentic nitrate. This should be allowed to dry. The labia should be kept separated to allow for free drainage; this is accomplished by placing a pledget of cotton or antiseptic lint saturated with 20 per cent argyrol solution against the urethral meatus between the swollen vulvar lips. After a few days, by following the above directions, the physician will be rewarded by seeing the discharge markedly diminished and the vulvar swelling lessened. The bowels may be moved by salines; *enemata are dangerous in the presence of gonorrheal discharges,* for gonorrheal proctitis is a most intractable complication. The urine may be rendered bland and non-irritating by the copious exhibition of pure water and milk, and by the administration of full doses of urotropin, or of urotropin with benzoate of soda. The patient should remain in bed while there is pain and bladder irritability; also for the space of, and for some days after, the menstrual period, as it is during this time that upward extension is most likely to occur. Autogenous vaccines seem to have some curative value.

Follicular Vulvitis.—In follicular vulvitis the inflammation is limited to the hair follicles and sebaceous glands about the vulva. Small red papules are scattered over the labia, and these become pustules with a hair in the apex of each. The surrounding tissues may be unaffected or may become more or less inflamed from the confluence of the lesions.

Irritating discharges, lack of cleanliness, parasites, and local irritation are the chief predisposing causes. The staphylococcus albus is the direct cause of the pustular formation; occasionally the condition occurs during pregnancy.

Symptoms.—The symptoms are the same as have already been described under simple vulvitis.

Treatment.—The vulvar hair should be clipped, and the vulvar surfaces carefully cleansed with Synol soap and water, rinsed with warm water and dried. The hairs in each pustule may be extracted with suitable forceps, and compresses saturated with alcohol 50 per cent, and boric acid solution, applied. Greater effect may be obtained by covering

the compresses with rubber tissue, which in turn is held in place with a T-bandage. The compress should be changed frequently. Rest in bed contributes to speedy recovery. When the pustules are few in number, after the hairs have been removed from each pustule, they may be painted with the tincture of iodin, which is allowed to dry, and the vulvar surfaces left uncovered.

Diabetic Vulvitis.—Diabetic vulvitis is caused by the decomposition of diabetic sugar loaded urine passing over the vulvar surfaces, which produces a dermatitis. The tissues of the vulva and inner surfaces of the thighs are of a reddish copper color and the skin is parchment-like, corrugated and dry with moist areas in the folds of the groin. The itching is constant and there may be local pain and tenderness. Eczematous changes frequently take place in the skin. The patient is generally poor in health from the diabetes and her disturbed rest.

TREATMENT.—The treatment is both general and local; the general management consists in a regulated diet and the adminstration of large doses of bicarbonate of soda. Locally alkaline vaginal douches always give the patient comfort. The pruritus may be relieved by ointments or dusting powders after the parts have been carefully cleansed with soap and water and the skin dried with absorbent cotton. An ointment such as the following may be applied and has given much relief:

℞

Ac. salicylici ..... gr. x
Ungt. petrolati ..... ℥ j

Or a skin protective of the following may be used:

℞

Zinci stearati ..... ℥ iss
Ac. carbolici ..... gr. v.
Glycerini ..... ℥ iss
Pulv. tragacan. ..... gr. xij
Aquae ..... ℥ j

Dusting powders of stearate of zinc and cornstarch blown on the surface may relieve the itching. Excoriations or abrasions of the skin should be painted with silver nitrate, gr. xx-℥ i, and then covered with ointment.

INFLAMMATION OF THE VAGINA

Vaginitis or colpitis is an inflammation of the vaginal mucosa: it may be *acute or chronic,* although it occurs most frequently in the latter form.

The histologic structure of the vaginal mucosa, covered as it is with several layers of pavement epithelium, together with the secretions found in the vagina, offers considerable resistance to bacterial invasion. The absence of vaginal glands further contributes to this resistance of the vaginal mucosa to the invasion of infecting bacteria. In the non-pregnant condition the vagina is kept moist by the secretions from the uterus and the cervix; but in pregnancy, owing to the increase in the pelvic circulation, the secretion is markedly increased. These secretions are bactericidal in their action, as has been shown by both Döderlein and Williams. Hence it is difficult for pyogenic organisms, which may gain entrance into the vagina, to live therein and cause inflammatory changes, unless the natural vaginal secretion is modified in character, or the resisting lining of pavement epithelium is impaired by trauma, thus diminishing the tissue resistance. Yet under certain conditions, the germicidal action of the vaginal secretion may be impaired or the development of pyococci increased, and thus an infection of the mucosa occurs.

In childhood or old age, or when the secretions from the cervix or uterus are increased and changed in character—as is the case at or near the menstrual period, after labor, or abortion, or when the uterus is prolapsed—the vaginal resistance is impaired: furthermore, when the vulvovaginal orifice is relaxed, the vagina becomes more sensitive to bacterial invasion. While vulvitis frequently occurs alone, vaginitis is usually complicated with vulvitis. There are four clinical varieties of vaginitis: (1) simple vaginitis, (2) gonorrheal or specific vaginitis, (3) granular vaginitis, (4) senile vaginitis. Occasionally, as a complication of the exanthemata, we may see diphtheritic vaginitis. In the chronic form it usually occurs in patches; it is seldom that the entire vaginal mucous membrane is involved.

Causes.—The causes are both predisposing and exciting. Under the former we may include anything which impairs the local resistance of the vagina to infection, such as mechanical or chemical irritation, youth, old age, feeble health, and such local and systematic conditions as may result in the desquamation of the vaginal epithelium. *The exciting causes are bacterial,* the gonococcus, the streptococcus, the staphylococcus, bacillus diphtheriae, and the colon bacillus being the chief etiological offenders.

The gonococcus of Neisser is especially active in the causation of the vulvovaginitis found in children. In the exanthemata, the vaginal mucosa participates in the general inflammatory involvement of the mucous membranes, while in puerperal vaginitis, the streptococcus and the pyogenic bacteria play an active part in the causation. Simple non-

specific vaginitis is commonly the result of irritating discharges, cauterants, and mechanical irritants.

Symptoms.—The symptoms of acute vaginitis vary with the severity of the involvement. There is commonly a feeling of heat and pain in the vagina, which may be dull or intense in character, a throbbing sensation in the perineum, backache, and a slight elevation in temperature at first. The vaginal mucosa is hot and dry, for the normal vaginal secretion is diminished; but in from twenty-four to forty-eight hours the vaginal mucosa becomes bathed in a thin, white mucous discharge, which soon becomes mucopurulent in character and runs over the external genitals. This causes intense vulvar irritation, frequently resulting in pruritus. The local symptoms are always more pronounced at the time of the menstrual period. On inspection the vaginal walls are red, swollen, and sensitive, and are bathed in a profuse purulent discharge; though as a rule the entire vaginal surface is not involved, the inflammation occurring in patches, separated from each other by healthy tissue. There are usually no urinary symptoms in simple vaginitis; this, however, is not true in the gonorrheal type.

Gonorrheal Vaginitis.—In the acute gonorrheal form a history of a suspicious intercourse is always suggestive; furthermore, the symptoms are more intense, while the discharge is more profuse, and of a purulent character, yellowish or greenish in appearance, and of a faintly acid or neutral reaction. No positive diagnosis should be made *unless the gonococcus can be demonstrated microscopically.* The urethra and the urethral glands are usually involved, so that frequent and burning urination is the rule. Gonorrhea of the vagina is usually secondary to an infection beginning elsewhere. On inspection, after wiping away the pus from the vestibule, fresh pus may be expressed from the ducts of Skene's and Bartholin's glands. The vaginal mucosal membrane is dark, red or bluish in color, is more or less thickened and studded with patches of erosion which bleed easily on contact. Moist vegetation may appear about the vulva, secreting a seropurulent fetid discharge.

In the chronic form there may be few or no symptoms, except vulva irritation and dyspareunia, due to the mucous discharge which excites some inflammatory reaction in the tissues about the introitus. Exacerbations may occur near the menstrual period when the gonococcus rapidly develops in the culture medium offered by the menstrual blood, and may be demonstrated in the discharges.

Granular Vaginitis.—Granular or papillary vaginitis is usually subacute or chronic; the granulations may result from gonorrheal infection, or from the congestion due to pregnancy. The papillae of the vagina

swell and become infiltrated with small cells, giving the mucous membrane a granular appearance. The granulations are hemispheric in shape, small in size, and scattered over the upper surface of the vagina and the cervix. The granular vagina and cervix of the pregnant woman is of common occurrence, especially in brunettes; it is seldom however associated with simple vaginitis.

Symptoms.—The symptoms are usually not severe; there is a sense of fullness and pressure in the vagina, and a sero- or mucopurulent discharge which, as it escapes over the external genitals, causes a vulvar pruritus. Exanthematous patches on the vulva are frequently present in this form of vaginal inflammation, while small areas of granulations are scattered over the vagina and cervix.

Senile Vaginitis.—Senile vaginitis or adhesive vaginitis is usually found in women past the menopause. It is characterized by the formation of adhesions due to the atrophic changes of old age, which result in defective nutrition and local destruction of the protective epithelium; this leads to infection and superficial ulceration. Hence, those portions of the vaginal wall which come in contact with each other after the epithelial covering has been destroyed are apt to become adherent.

Symptoms.—The symptoms which cause the patient to seek medical advice are a thin, serous, acrid, vaginal discharge, which is seldom profuse, but is frequently blood stained; and a burning sensation in the vagina, or a feeling of weight in the pelvis, or marked irritation, such as a pruritus or dermatitis of the external genitals. The marital relations may be painful or impossible, owing to the atrophic changes in the introitus and in the vaginal walls. On inspection the mucous membrane is found smooth, while various sized ecchymotic spots and superficial ulcerations are seen scattered over its mucous surface, which is moistened with a scanty serous secretion. Digital exploration may detect adhesions between the cervix and the vagina or between the opposing vaginal walls.

Prognosis of Vaginitis.—The prognosis of simple vaginitis is usually good when proper treatment is instituted. In the gonorrheal variety the prognosis is fair as to the immediate condition, but must always be guarded, for the infection may remain latent and become active at some later date. Under favorable circumstances it has a tendency to extend to the endometrium, tubes, ovaries, and pelvic peritoneum. Chronic vaginitis is often intractable, recurring from time to time with intense pruritus. In the diphtheritic cases and those due to active cauterants or irritants (as an ill fitting pessary) atresia of the vaginal tube may result from sloughing of the mucosa and adhesions of the opposing walls, or occlusion of the vagina may ensue from cicatricial contraction. In the

granular form the condition often disappears spontaneously at the end of gestation, although, when of gonorrheal origin, granulations may be found in the vaginal vault years after the original infection has been cured. Senile cases are usually intractable and can be made comfortable only by persistent and frequently repeated treatment.

Treatment.—The treatment in acute vaginitis should include: (1) rest in bed and posture, (2) cleanliness, (3) relief of pain, (4) dietary regulation. Rest in bed is essential during the acute stage, even in mild cases, and postural drainage favors local tissue recovery. The bowels may be regulated with saline laxatives, or laxative waters. An alkaline, salt free, or non-stimulating diet contributes to a happy termination. Codein, ½ to 1 gr. repeated every three to six hours, may be used to relieve the pain. Any posture that is comfortable to the patient and *insures free vaginal drainage is permissible.* Cleanliness is insured not only by proper drainage, but by the use of copious saline irrigation given several times a day with the douche can or bag at low elevation.

In the subacute condition, silver nitrate in two per cent solution can be used to advantage. Warm Fuller's earth may be packed in the vagina through a Ferguson speculum and held in contact with the inflamed surface by a wool tampon; this should be removed in twenty-four hours and a cleansing douche given.

In the acute gonorrheal variety the general principle of rest, posture and cleanliness, and dietary restrictions as previously outlined should be carried out. One change however is to be observed: *no douches or irrigations should be given during the acute stage.* If more active treatment is desired, argyrol 10-25 per cent solution may be used; this is best applied in the following manner: with the patient in the dorsal position and the hips well elevated, the labia are carefully separated, and from a feeding cup the argyrol solution is poured into the vagina. The amount to be used should be sufficient to insure moderate distension, reaching all parts of the vagina. The patient is then returned to the Fowler position and the fluid allowed to escape. Care should be taken that proper pads are placed under the patient to absorb the excess of argyrol.

Once the acute stage has passed, more active treatment may be instituted in the form of potassium permanganate douches, 1:4000, and the topical application of argyrol, 25 per cent, and silver nitrate, 2-4 per cent, to the entire vaginal surface.

Treatment of Senile Vaginitis.—Senile vaginitis is frequently but a local manifestation of the atrophic changes which take place in women in the postclimacteric period. To how great an extent the endocrine glands enter into the etiology of this condition is hard to say, but

unquestionably they play a part. Clinically ovarian extract in doses of 2-5 grains three times a day often seems to help. The use of other endocrines, of course, depends largely on the patient's general condition, and this must be determined by the physician in attendance. There is no question that better results are obtained by the routine use of ovarian and thyroid extracts in combination with local measures, than by topical applications alone. These patients should be encouraged in the liberal use of alkalies, such as the internal administration of sodium and magnesia carbonate, while the topical application of silver nitrate, in strengths of 2-10 per cent, is often of assistance.

ENDOCERVICITIS

Endocervicitis is an inflammation of the intracervical mucous membrane, and may be either an acute or chronic, and may occur either as a primary or secondary infection. It is primary when the infecting agent directly invades the cervical mucosa, and secondary when the infection extends upward from the vagina or downward through the uterus.

Acute Endocervicitis.—The acute form is due to direct invasion of the mucosa by septic or specific bacteria. When of septic origin, cervicitis is but a part of the general infection of the uterovaginal tract. It never occurs as a pathologic entity. On the other hand, the gonococcus may cause an acute primary endocervicitis without involving the adjacent tissues, or be checked at the internal os and the inflammation pass into the subacute or chronic stage, involving the glandular crypts of the cervix and producing a chronic glandular cervicitis. The infection may remain latent for long periods, unless it is carried on into the uterus by meddlesome instrumentation. Furthermore, because of the anatomic relations of the cervix, it must necessarily participate in the acute infections of the uterus and the vagina; hence, acute cervicitis need not be considered clinically as a pathologic entity, for both its symptomatology and treatment are those of the associated lesions.

Chronic Endocervicitis.—Chronic endocervicitis is a low grade inflammation of the cervical mucosa. This inflammatory process may be confined to the mucosa of the cervical canal, or may extend into the deeper tissues of the cervix, producing a cervicitis.

Endocervicitis is the most prevalent of all gynecological disorders. Fully 85 per cent of all women, single or married, have infected cervices. It is the same condition described as "cervical erosion, cervical catarrh, or cervical endometritis" by the older writers, but strange to say, they did not understand the pathology and wholly failed to appreciate the infective origin of the disease. Their methods of treatment therefore, as

might be expected, were palliative and not curative, hence in the majority of instances were tedious and unsuccessful.

In both structure and function, the cervical mucosa differs widely from the mucosa of the corpus uteri. The cervical canal, lined with its mucosa, simply acts as a passive communicating channel between the vagina, which is always the habitat of many and various bacteria, and the uterine cavity. On the other hand, the corporeal endometrium is constantly passing through such active changes as are essential to the function of menstruation and deciduation. The cervical mucosa *contains large numbers of deep, penetrating racemose glands and evinces a marked susceptibility to infection;* while, according to Curtis, the normal corporeal mucosa is practically immune to infection.

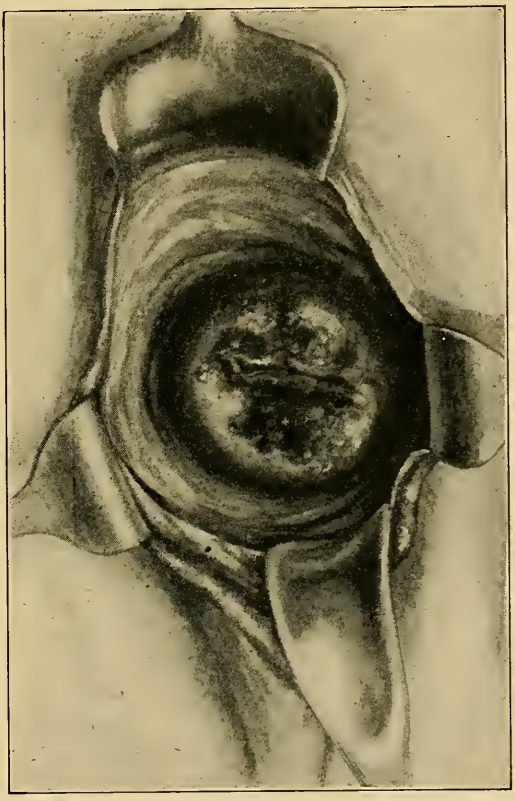

Fig. 3.—Cystic Cervicitis Resulting from Infection of a Cervix Laceration.

Etiology. — The organisms most commonly found to be the infecting agents in endocervicitis are the gonococcus, staphylococcus, streptococcus and colon bacillus. The gonococcus and the staphylococcus are by far the most prevalent. *Trauma,* lacerations during childbirth or the result of instrumental delivery, cauterization, or the constant irritation of a stem pessary, may open up avenues of entrance for infection, and are thus predisposing factors in the production of chronic cervical inflammation. *But trauma is only a contributory cause,* for as Sturmdorf so aptly says, "the dominating pathologic factor that determines the morbidity of a cervical laceration is not the extent of the tear, *but the incidence of its infection.* Such an infection does not remain limited to the lacerated area, but sooner or later involves the entire endocervical mucosa and the subjacent tissue from the internal os."

In children, vulvovaginitis or the exanthemata, particularly diphtheria and scarlet fever, and the general debilitating diseases may produce in the cervix a lowered tissue resistance, increasing its susceptibility to mixed infections. Hess says that "we must regard the average gonorrheal vulvovaginitis in children as involving the cervix rather than the vagina, and must consider the infection as a cervicitis rather than a vaginitis."

As we have already stated, acute endocervicitis and cervicitis occur in the course of acute gonorrheal and puerperal infections of the uterus, but are seldom recognized as definite pathological entities, as their symptomatology is masked by the more extensive lesion in the uterus.

Pathology.—The mucosa of the cervix, when it is a seat of chronic infection, becomes swollen and everted, while the mucosa of the portio about the external os presents a circumscribed area of glandular proliferation. The columnar epithelium covering the mucosa of the cervical canal, under the constant stimulation of infection, rapidly proliferates and actually *pushes itself out on the vaginal aspect of the cervical rim,* replacing the stratified epithelium which is normally present in this situation, producing the so-called *erosion.* This erosion or "red area" about the external os, therefore, is not ulceration, *but is actually a new cell formation, an overgrowth of lymphoid tissue,* which may, under certain conditions, become malignant. The continued congestion incident to such a condition produces a hypersecretion of mucus from the gland structure, and a hypertrophy and hyperplasia of cervical connective tissue. Sooner or later, owing to the tissue hyperplasia, the crypts of the cervical glands become occluded and the secretion is retained, thus forming retention cysts, the so-called nabothian cysts, so commonly found in this region of the cervix.

These cyst formations are produced partly by the hyperplasia of the periglandular connective tissue, encroaching upon the lumen of the gland duct, and partly by the overproduction of thick viscid mucus which may plug the outlet ducts; or, during the process of cure, or even of attempted healing, the squamous epithelium may grow out over the erosion and actually "choke" or cover over the gland openings. This cystic condition naturally increases the bulk of the already hypertrophied cervix and thereby further interferes with its *circulation and muscular contraction.* It is this hyperplasia which causes, by interference with the cervical circulation, the premenstrual and the postmenstrual metrorrhagia, so frequently met with in the general cyst formation of the cervix.

Microscopically we find the evidences of a chronic inflammation, or on the other hand, there may be very little inflammatory change in the stroma, and except for a preponderance of gland tissue, the section may

appear almost as normal cervical tissue. Section of a distended nabothian follicle may show as a large clear space lined with columnar epithelium and filled with mucus. Surrounding this, there may be the signs of a subacute inflammation, as shown by round cell infiltration, congestion, edema, dilated lymph spaces, etc. In the musculature nearest the cervical mucosa there may be found small inflammatory foci or even small multiple abscess formations.

Serial sections made from the excised cones of cervical tissue similar to those shown in figure 4 show erosions, chronic inflammation, cystic changes, etc., as described, in varying degrees of intensity. Beginning at the distal or portial end, we find marked inflammatory changes, but as we approach the proximal extremity, i.e., near the internal os, these changes are either barely perceptible or are entirely absent, showing the efficacy of the surgical procedure to be described under treatment, in so far as the removal of the infected area is concerned.

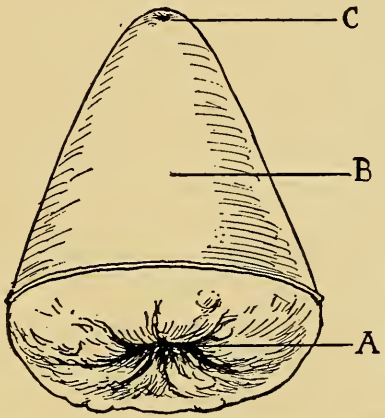

FIG. 4.—CONE TAKEN FROM INFECTED CERVIX, SHOWING POINTS FROM WHICH SERIAL SECTIONS WERE MADE.

CLINICAL COURSE.—The clinical course of chronic endocervicitis is *slow and insidious* and shows little or no tendency toward a spontaneous cure —the symptoms are variable. In the milder cases, where the infection is not sufficiently virulent to be progressive, *cervical leukorrhea* of a mucoid or mucopurulent character, which may be scanty or profuse, *is the only symptom complained of;* very simple to describe, but often extremely invidious to the patient and well nigh invincible to the physician. When the infection extends to the deeper structure of the cervix, *the uterus may become involved.* This is brought about by an ascending lymphangitis between the uterine muscle bundles. Such a condition naturally excites a tissue reaction with a deposition of inflammatory products between the muscle fibers; this in turn interferes with the rhythmic contraction of the uterine muscle fibers, and allows a resulting circulatory stasis, thus interfering with drainage and favoring chronic infection and involvement of the parametrial tissues. The fallopian tubes later become involved through a parametritis; a perisalpingitis, peritonitis, and peri-ovaritis may result. Abdominal section on several hundred cases of chronic endocervicitis of long standing, has never failed to show some evidence of the ex-

tension of the infection to the peritubular structures and the ovaries. In the presence of such a circulatory and lymphatic stasis, the pelvic structures are more susceptible to new infection and less resistant to old infection.

Symptoms.—Chronic endocervicitis is *always attended by some degree of posterior cellulitis in the uterosacral ligaments.* This produces the premenstrual and comenstrual backache as well as the dyspareunia so frequently complained of; *however, the prominent symptom in most cases is leukorrhea.* This discharge is intermenstrual, is mucoid in character, and is usually profuse and viscid; it may be translucent or opaque, or curdy, or it may be purulent when the inflammation is suppurative. It is always *more abundant just before and after the menstrual periods.* The patient may also suffer from metrorrhagia, resulting from the circulatory stasis due to the intermuscular lymphangitis, which has impaired the contractile power of the uterine muscle. The lumbosacral pain is due to the traction of the cervix on the thickened tender uterosacral ligaments. Some patients may complain of no symptoms, *though a speculum examination will always reveal the erosion, the cystic hyperplasia, and the presence of a cervical discharge.*

Fig. 5.—Gross Appearance of Excised Cone, Showing Muscle Tissue Replaced by Fibrous Tissue.

Sterility in women with apparently normal cervices, which may or may not be anteflexed, is in the vast majority of cases due to the *existence of a chronic endocervicitis,* dating either from childhood or from an infection contracted soon after marriage. Such an infection need not be gonorrheal, for we know that staphylococci from the husband's prostate gland are often sufficiently virulent to infect the cervical mucosa of his

wife, and produce an intractable endocervicitis, even though his last debauch was twenty years previous to his marriage.

Reynolds, of Boston, has demonstrated that a spermatozoön, no matter how vigorous, cannot long withstand the strain of progress through the thick tenacious mucus and clumps of pus cells that plug the canal of a diseased cervix. Furthermore, the cervical infection following upon lacerations of the cervix in those women who have borne one child, but have subsequently remained barren, must be recognized as a very important factor in the production of their continued sterility.

Differential Diagnosis.—Chronic endocervicitis and cervicitis have to be differentiated from malignant disease of the cervix and cervical tuberculosis. In cancer of the portio vaginalis in the initial stage the affected lip is nodular and indurated. The nodules are glazed and bluish white and the mucosa may be unbroken, or the mucosa may be eroded, in which case the eroded area is always friable, bleeds easily on manipulation, and the surrounding cervical tissue is hard and infiltrated. In chronic cervical inflammation, the everted area is covered with thick tenacious mucus or mucopus. In cancer the secretion loses its mucoid character and the erosion secretes a serous, *serosanguineous, or seropurulent* discharge. A histologic examination will make the diagnosis positive.

Primary tuberculosis of the cervix is very rare; it may appear in three pathological forms: (a) the ulcerative, (b) the hyperplastic, (c) the miliary (Beyea). The tubercular ulcer of the cervix is sharply cut, irregular, undermined, and usually commences near the external os and spreads over the portio vaginalis and up the canal. The edges may be covered with necrotic material, while the floor is soft and there is an absence of induration. The base is uneven and tends to heal by cicatrization. The diagnosis is based upon the microscopical findings, which will show the presence of giant cells, tubercles, and tubercle bacilli, in and about the ulcer.

In the hyperplastic form the cervix is grossly enlarged and hard, simulating chronic cervicitis, and the tubercles are within the muscle bundles, or surrounded by masses of protective connective tissue in the stroma.

Miliary tuberculosis of the cervix has seldom been recognized. The miliary type of lesion is an early stage of ulceration; granulating tubercles protrude, these may be isolated or confluent. A positive diagnosis can only be made by the aid of the microscope, the clinical history, and in finding tuberculosis elsewhere in the body.

Syphilitic ulceration of the cervix may occur, but it is extremely rare.

While a chancre of the cervix may be found on either the anterior or the posterior lips, it may appear as a single ulcer, or be multiple. The characteristics of chancre of the cervix do not differ from those of chancre in other parts of the genital tract. The diagnosis depends upon the presence of the characteristic lesion, a positive Wassermann, together with other evidences of luetic infection. Microscopic examination of the diseased tissue and finding of spirochetes will confirm the diagnosis.

Prognosis.—*The prognosis in chronic endocervicitis must be guarded.* When the infection is slight and the coincident inflammation mild, and proper treatment is instituted early, complete recovery may take place. On the other hand, when the infection is virulent and the inflammatory changes within the cervical tissue are marked, the cure is difficult, as there are many associated lesions. The cervical glands, because of their histologic structure, may harbor infecting organisms for years. These glands are situated in a tissue that is richly supplied with lymphatic drainage; and these lymphatic channels are connected with lymph channels of the uterus, which in turn communicate with lymphatic chains in the parametrial tissues and in the uterosacral ligaments. Hence, it will be seen that the associated lesions, as metritis, parametritis, and uterosacral cellulitis, are progressive, and not until the focal infection is cured, can these *associated lesions which actually produce the symptoms* of leukorrhea, menorrhagia, metrorrhagia, and lumbosacral backache be improved.

Long continued cervical inflammation may be considered a *prodrome of cervical cancer,* for it is but a step from the extreme cell proliferation with an orderly arrangement that occurs in hyperplastic endocervicitis and cervicitis, to the disorderly arrangement of embryonal cells found in cancer.

Treatment.—The treatment of chronic endocervicitis is palliative or operative. In considering the treatment of any infective process, we should in the usual course of events consider first, the treatment during the acute stage, and second, that of the chronic; but unfortunately the acute stage of endocervicitis as a pathological entity, is seldom recognized, and for that reason the disease becomes subacute or chronic, with its resulting pathology, long before the patient seeks medical advice.

In acute endocervicitis of gonorrheal origin rest, postural drainage, and local cleanliness, are the fundamentals of treatment. Alkaline douches of warm borax or soda bicarbonate solution, given at a low elevation, will dissolve and wash away much of the profuse purulent or mucopurulent discharge. Great care must be exercised to avoid producing any trauma and thus disturbing nature's effort to arrest the

infective process by tissue reaction. The same general principles of rest, postural drainage and cleanliness likewise apply to postabortal and puerperal endocervicitis, but here, however, the endocervicitis is only a part of the endometrial infection, and as the internal os, which in the uterus is the natural barrier to the upward extension of infection, is never closed in the acute stage of a postabortal endometritis, *direct local measures should be avoided.*

The palliative treatment of chronic erosions of the cervix, as commonly given in office practice, is often more harmful than beneficial, for the reason that many practitioners do not appreciate the pathology or the importance of the incidence of infection in treating these eroded surfaces. Simple non-infected erosions will usually clear up with improvement of the uterine circulation. When complicated by retroversion this may be done by reposition of the uterus and its retention in the normal position with a properly fitted pessary; and a few applications of Churchill's tincture of iodin, or a single application of pure carbolic acid to the eroded surface, made after the mucus has been removed by the application of a paste of bicarbonate of soda mixed with peroxid of hydrogen to the mucus covered area. In the non-parous married woman sexual abstinence will do much toward improvement of the uterine circulation and thus favor healing of the erosions.

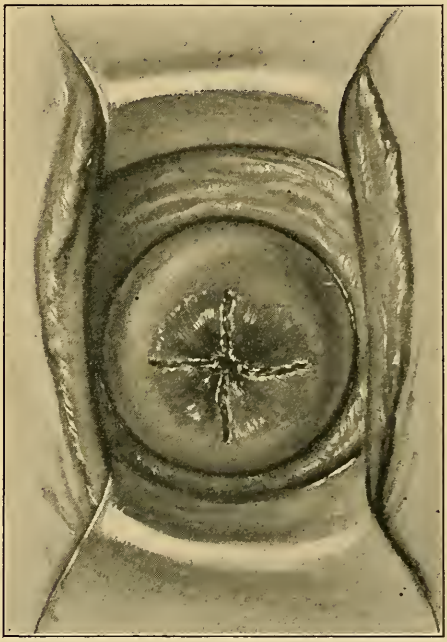

FIG. 6.—CHRONIC ENDOCERVICITIS TREATED BY LINEAR CAUTERIZATION.

If these simple measures fail, the dry treatment suggested by Gellhorn and Curtis will frequently result in a cure, provided, however, the deeper structures are not involved. This treatment consists in thoroughly cleansing the vagina and cervix with a hot alkaline douche. The cervix is then exposed through a Ferguson speculum and the cervical canal and the erosion with the least possible trauma is cleared of mucus by the application of peroxid paste. After the mucus has thus been cleared away, hot Fuller's earth is packed about the cervix and kept in place by filling the vagina with a gauze tampon. The gauze is removed

Fig. 7.—Same Cervix After Linear Scars Made by Cautery are Healed.

in twenty-four hours and the patient takes a hot alkaline vaginal douche. This treatment is repeated every other day, and in those instances where the infection has remained in the superficial tissues, the erosion and leukorrhea are in a large proportion of the cases cured.

In the more extensive erosions associated with moderate degrees of cervical laceration and ectropion, the eroded areas may be healed by linear cauterization after the method of Hunner and Russell. The technic of this procedure is as follows: With the patient in the Sims position and the cervix exposed by a Sims speculum, the anterior lip of the cervix is grasped with double tenaculum and held in position, while the cervical canal is cleared of mucus. The fine point of the actual cautery knife, cold, is then introduced into the canal to a point just beyond the uppermost limit of the ectropion. The current is then turned on and a linear incision, going through the mucosa into the underlying myometrium, to the depth of about one-eighth to one-fourth of an inch, is made. The cervix is then painted over with iodin, and the patient is told to return in two weeks, when another similar incision is made on the opposite aspect of the canal. Two weeks later a third incision is made midway between the first and second, and finally we make a fourth incision opposite the third (Fig. 6). In all four, linear cauterizations are made, and as the resulting ulcers heal and the tissue retracts, the ectropion disappears, provided the treatment has been successful. There is at least one valid

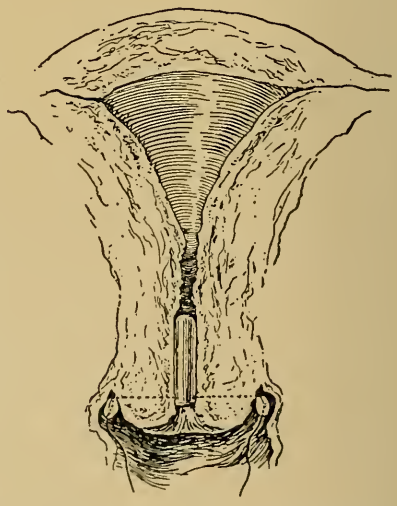

Fig. 8.—Twenty-five Milligrams of Radium in a Brass Filter Placed in the Cervix.

objection to the employment of this treatment, and that is, that by the time this treatment is instituted, many of the cervical glands have already become infected and the resulting linear cicatrix may plug the lumena of the ducts and leave infected glands buried in the deeper structures, which may later form cysts.

Another form of treatment that has been recommended is the thorough curettage of the cervical canal and that part of the everted mucous

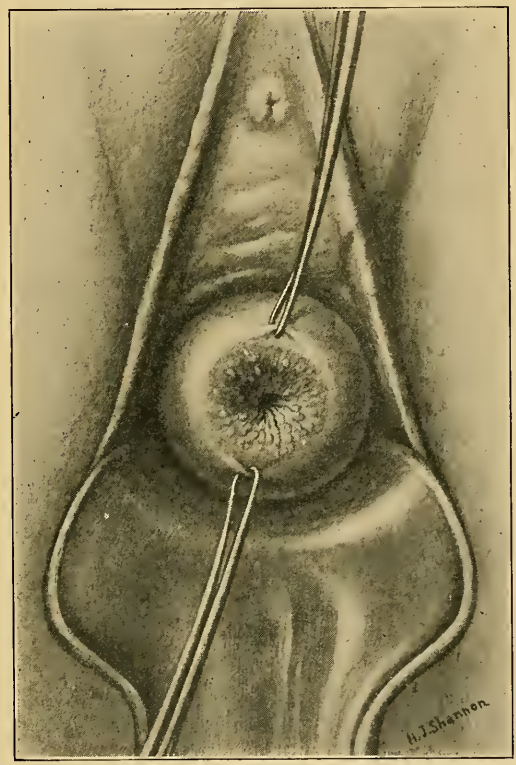

FIG. 9.—TENACULA ON THE ANTERIOR AND POSTERIOR LIPS OF THE CERVIX WITHIN THE MARGIN OF THE EROSION.

membrane surrounding the external os with a specially devised small, sharp curet. This method, we believe, is open to the same criticism as the Hunner cauterization method. Curettage can never remove all the gland tissue of the cervical mucosa, and wherever the gland tissue remains undisturbed, the infection remains unmolested. *The infecting organisms inhabit the most remote branches of the deepest glands in the cervical mucosa,* and therefore, the futility of eradicating all the infective foci by curettage is at once apparent.

After giving all these methods a fair trial, we feel that, aside from

the simple erosions of limited area which are not infected, palliative treatment does not cure endocervicitis.

Recently, at the suggestion of Curtis, we have used radium for the cure of leukorrhea and cervical erosion, when the bulk of the cervix has been increased by tissue hyperplasia. With the cervix exposed and cleared of mucus, a capsule containing 25 milligrams of radium enclosed in the proper filter is introduced into the cervical canal, and held

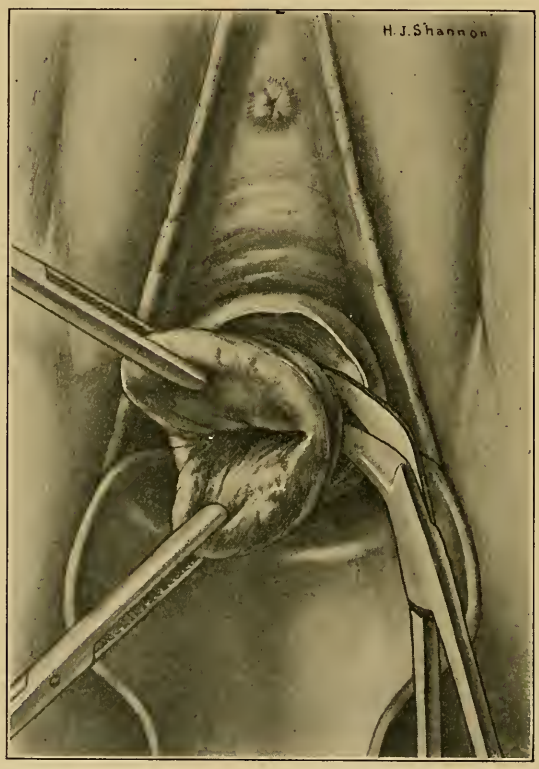

Fig. 10.—The Same Procedure May Be Used in Infected Lacerations.

in place with a very narrow gauze packing, or by placing a stitch in the portio which passes through both lips and the ring of the capsule (Fig. 8). The application is kept in the cervix for eight hours and then removed. Within a few weeks the erosion is healed, the leukorrhea is checked, and the bulk of the cervix materially reduced. Our experience, however, is too limited for us to draw any definite conclusions, except to advise its further trial.

Operative Treatment.—Sturmdorf, in his very excellent work, has called attention to the pathology resulting from chronic endocervicitis,

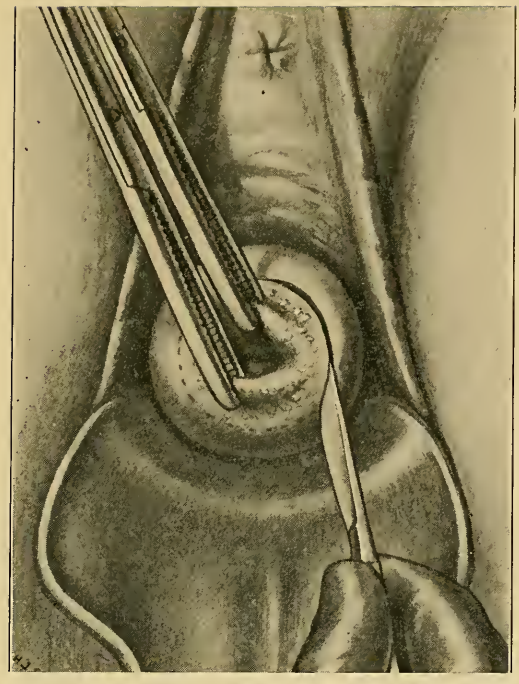

Fig. 11.—Incision Circling the Limits of the Erosion.

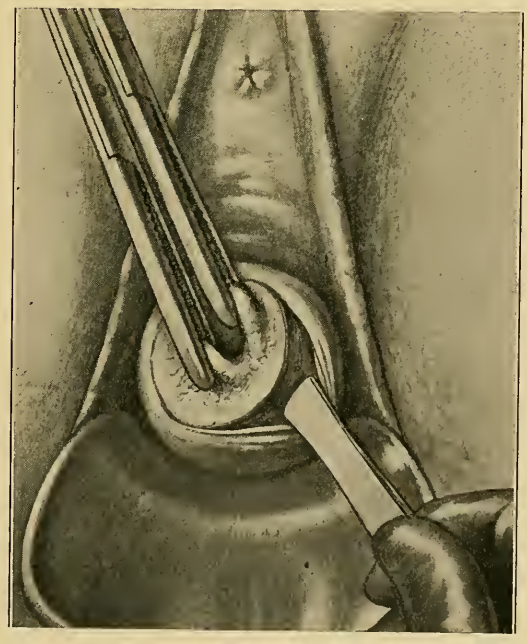

Fig. 12.—The Portial Mucosa Is Then Pushed Back to Make the Cuff.

and has suggested excision of the infected area by a method which preserves the greater part of the cervical musculature.

During the past three years, at the Long Island College Hospital, we have treated all cases of extensive chronic endocervicitis with the associated tissue hyperplasia already described, which have not yielded to some of the palliative measures, by the surgical method of Sturmdorf.

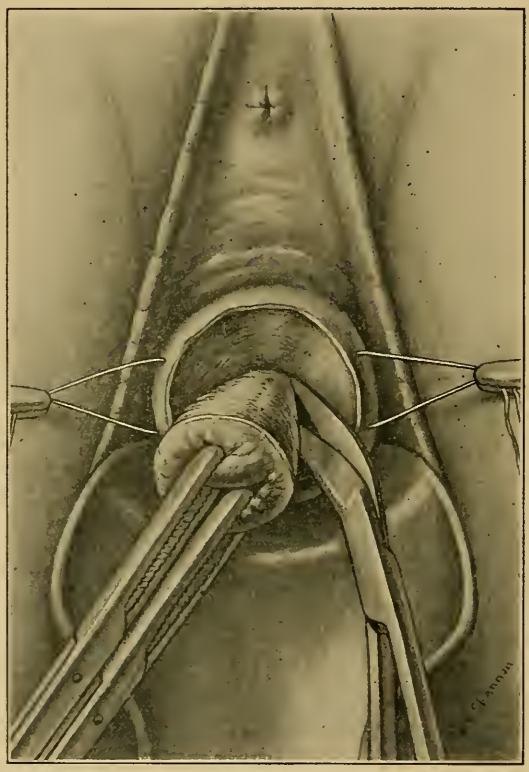

Fig. 13.—With Sharp Pointed Emmet's Scissors the Glandular Portion of the Cervix Is Coned Out.

The technic of this procedure with certain modifications which we believe are advantageous, may be described as follows:

With the patient anesthetized and placed in the lithotomy position, and the vulva, vagina, and cervix prepared for operation, the anterior and posterior lips of the cervix within the margin of the erosion are grasped with Jacob's forceps and the cervix pulled down. An incision is then made through the mucous membrane of the portio vaginalis encircling the limits of the eroded area (Fig. 11). The portial mucosa is now pushed back for a distance of three fourths of a centimeter, or more if necessary, to secure a sufficient cuff of the portial mucous mem-

brane to completely cover over the excised area of cervical tissue (Fig. 12). Then, with a sharp pointed pair of Emmet's scissors, while traction is being made on the cervix, the entire granular area surrounding the cervical canal is "coned out." The conical excision should extend up to or just below the internal os (Fig. 13). Great care must be exercised to preserve as much as possible of the muscle tissue, and still to remove all of the infected area. If too much cervical tissue is removed,

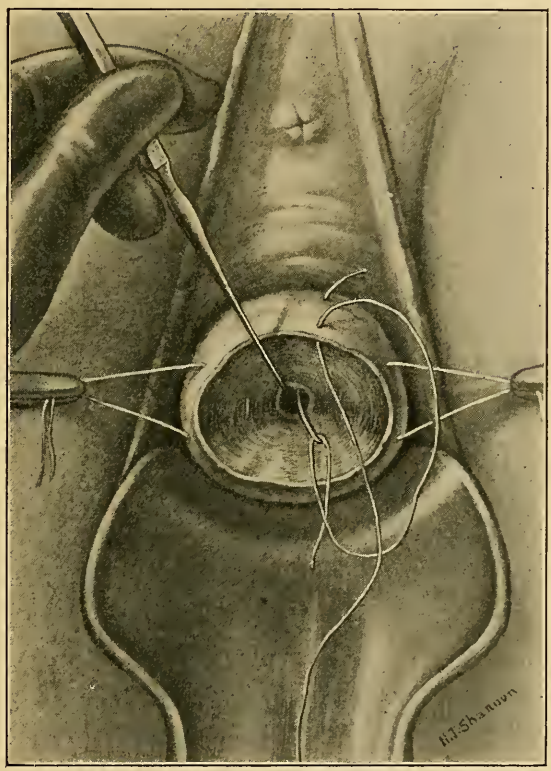

FIG. 14.—A SINGLE TENACULUM IS INTRODUCED INTO THE UTERINE CANAL AND THE TWO CATGUT SUTURES ARE HELD TAUT. THIS BRINGS THE UTERUS DOWN NEARER THE VULVA.

the operation becomes a high amputation, which for obvious reasons should be avoided. If, on the other hand, not enough tissue is removed, infected glands are left behind and the purpose of the whole operation is defeated. By keeping well within the muscle rim, the hemorrhage is inconsiderable, as the blood supply is distal to the line of excision.

Before removing the conical plug which is now attached by only a small rim of cervix tissue and cervical mucosa, two sutures of no. 2 chromic catgut on a full curved cervical needle are placed through the previously liberated flap of vaginal mucous membrane, to either side

of the partially excised cone. Each suture should include a good bite of cervical tissue (Fig. 14). When these sutures are drawn taut, the

FIG. 15.—SAGITTAL SECTION OF THE INVERTING STITCH.

operative field is brought well down toward the vulva. The cervical canal at the utmost limit of the cone is now opened and a single tenaculum is introduced into the canal (Fig. 14) for the purpose of further bringing the cervical stump down nearer the vulva. When this is done the remainder of the cone is excised. The vaginal flap originally liberated at the beginning of the operation is now inverted into the hollowed out shell of the cervix by the double inverting stitch of Sturmdorf. This maneuver brings the cuff of vaginal mucosa into contact with the cervical mucosa and thus completely covers all the denuded areas, and furthermore, substitutes healthy vaginal mucosa as a lining to the canal for the infected cervical mucosa which has been removed. This stitch is of silkworm gut and, when tied, not only coaptates the vaginal mucosa to the cervical mucosa, but controls bleeding from the tissues through which it passes (Fig.

FIG. 16.—WHEN THIS IS DRAWN TAUT THE PORTIAL MUCOSA IS IN CONTACT WITH THE MUCOSA OF THE CANAL.

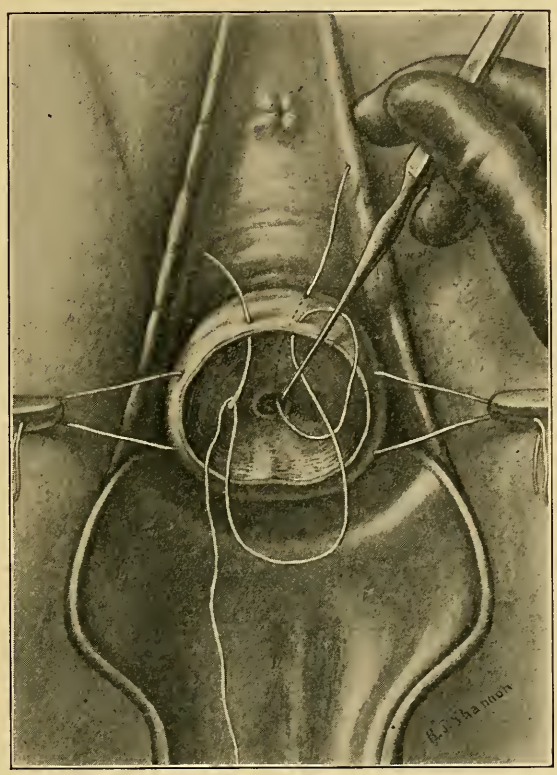

FIG. 17.—A SILKWORM GUT STITCH IS NEXT PASSED, AS PER DIAGRAM, THROUGH THE PORTIAL MUCOSA AND INTO THE CANAL.

17). This suture is left *in situ* for three or four weeks, or until the patient returns for her follow up examination, after her discharge from the hospital. The chromic gut sutures to either side, which have thus far acted as traction sutures, are now tied, bringing the gaping outer angles of the "coned incision" together, and completely controlling hemorrhage. With all sutures tied, six in number (Fig. 18), the operation is complete, and a small strip of iodoform gauze drain is placed against the cervical stump, which is removed in twenty-four hours.

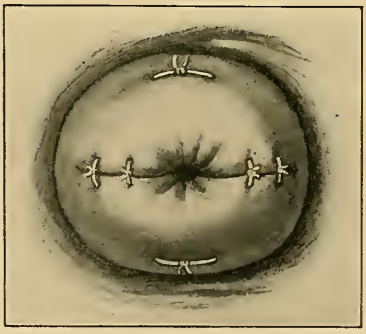

FIG. 18.—WHEN ALL STITCHES ARE TIED THE EROSION IS REPLACED WITH VAGINAL MUCOSA.

Objection has been raised to this procedure on the ground that it is too extensive for such a simple lesion, and that it may cause cervical dystocia during dilatation in subsequent labors. We have done this operation

for a sufficient time to form some definite conclusions as to its value. In the first place, it cures the leukorrhea and hence improves the probability of conception; second, a tracheloplasty of this type does not interfere with normal dilatation during labor; third, relief of the focal infection cures the posterior parametritis in the uterosacral ligaments, and cures the lumbosacral backache which is such a prominent symptom in chronic endocervicitis.

CHAPTER II

GONORRHEA

General considerations regarding gonorrhea—Frequency of gonorrheal vulvitis—Condyloma acuminata—Urethritis and bartholinitis—Gonorrheal vaginitis—Cervical gonorrhea—Gonorrheal endometritis—Histology—Differential diagnosis—Histology of chronic endometritis—Pathology and histology of gonorrheal metritis—Gonorrheal inflammation of the fallopian tubes—Characteristics peculiar to gonorrheal infection—Microscopic appearances of gonorrheal salpingitis—Pyosalpinx—Gonorrheal inflammation of the tubes and ovaries—Acute pelvic inflammation—Chronic pelvic inflammation—Treatment of gonorrheal infections—Acute specific urethritis—Acute specific vaginitis.

General Considerations.—The essential cause of gonorrhea is the gonococcus of Neisser (discovered in 1897). The gonococcus is an intracellular diplococcus of a double "biscuit shape." The two halves are separated by a narrow open space. It appears in groups of four, or multiples of four. It is small, non-motile, and does not have spores. The gonococcus is stained with methylene blue, but does not take the gram stain. It is common to find groups of these cocci within pus cells and at the margin of epithelial cells. They are never arranged in chains. The gonococcus may be positively identified by the microscope. By observing the following rules: 1, the prepared microscopical specimen shows the presence of a kidney or biscuit shaped diplococcus in groups of two or four, or multiples of four; 2, the situation of the coccus is within the body of the pus cell, though other bacteria may also be located intracellularly; 3, gonococci do not take the gram stain; 4, the gonococcus will not grow on gelatin or agar. Not only is the bacteriological diagnosis of gonorrhea of the highest medical and medicolegal importance, but bacteriologic examination is of considerable prognostic value, as the number of gonococci is usually in direct ratio to the acuteness and virulence of the infection. Furthermore, no case of gonorrhea should ever be discharged as cured until repeated examinations, *particularly on the day following the cessation of menstruation,* have failed to demonstrate the presence of the gonococcus.

While it is easy to confirm the clinical diagnosis of gonorrhea with the miscroscope in acute infections, in the chronic stage the finding and recognition of the gonococcus is frequently exceedingly difficult, owing

to some of the following conditions which are constant complications in chronic infection: 1, there are commonly but a few gonococci present, and these frequently lie deep in the gland crypts, only appearing during exacerbations of the local inflammation, yet they retain their infective qualities; 2, in chronic inflammations, not only are the gonococci few

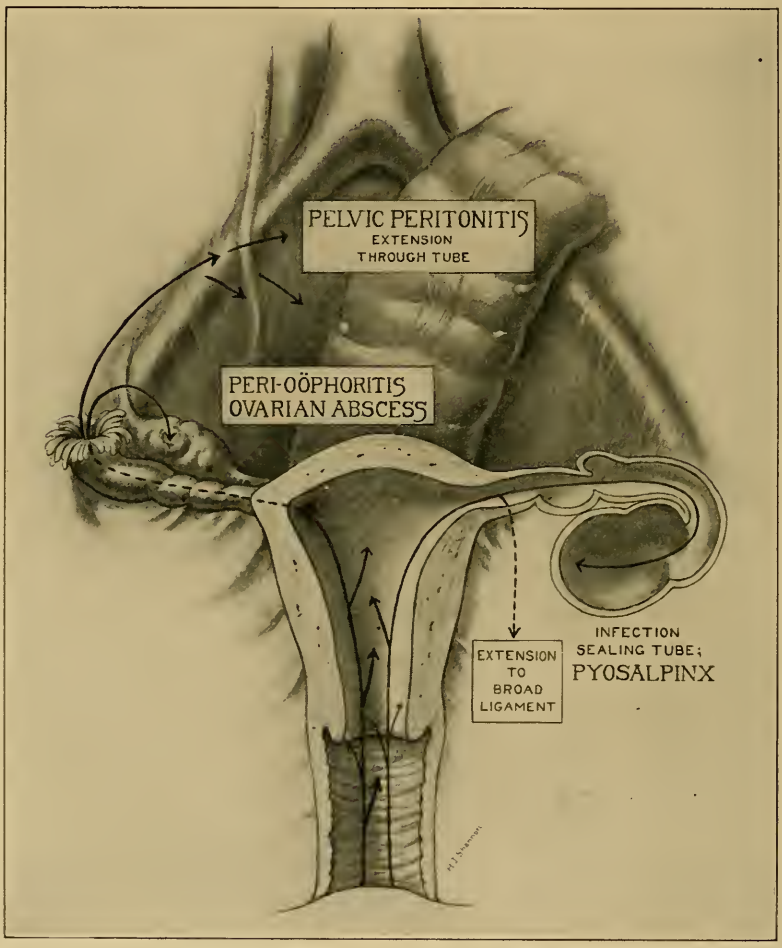

FIG. 19.—DIAGRAMMATIC DRAWING, SHOWING THE COURSE OF GONORRHEAL INFECTION THROUGH THE UTERUS TO THE PERITONEUM.

and far between, but there are also millions of other bacteria present, which make the recognition of the gonococci lying between and mixed up with them a most difficult task; 3, among urethral parasites there are found occasionally diplococci which show a marked similarity to the gonococcus, so that to decide whether the diplococcus found is a genuine gonococcus, sometimes presents great difficulties. Quoting from Neisser,

"it has been asserted by some authorities that gonococci change in regard to their forms, and that they can assume quite uncharacteristic appearances of degeneration without losing thereby their capacity for multiplication and their virulence, making it in this way possible for gonorrhea producing bacteria to pass unrecognized on account of the absence of all of their morphological peculiarities." Neisser says he has never been able, so far, to satisfy himself about the existence of such forms of degeneration; 4, the scanty number of gonococci present in a genital tract are not always accessible for examination, as they may be in hidden recesses, for the urogenital tract in the female is provided with many lurking places, such as the glands of the cervix, Skene's tubules, Bartholin's glands, and the vulvar pockets, in which the gonococci for many months or even years may maintain a subdued existence, yet capable of producing relapses when alcoholic or sexual excesses establish suitable conditions, and may thus retain their infectivity when brought in contact with virgin soil. In these chronic cases, where the organisms cannot be isolated, the complement fixation test offers a means of establishing the presence or absence of the gonococcus.

The complement fixation test is particularly valuable in the diagnosis of chronic gonorrhea, for Schwartz and McNeel state that a positive reaction can rarely be obtained before the fourth week of the disease; and the reaction persists for seven or eight weeks after the focal infection is cured, *hence a negative test in a patient who has previously had a positive reaction is good evidence that the disease is cured.*

Diagnostic vaccination by the subcutaneous injection of dead gonococci in persons suffering from gonorrheal infection is followed by a local and general reaction. This reaction consists of an area of swelling, redness, and tenderness at the site of the inoculation, and an exacerbation of the symptoms in the pelvis or joints, increased malaise, and sometimes fever. According to Sternberg, diagnostic vaccination is of much practical and contributory value in establishing the diagnosis of gonorrhea, when the organisms cannot be found in the discharges.

The gonococcus is not capable of infecting the vulvar and vaginal surfaces during sexual maturity, when these surfaces are covered with intact squamous stratified epithelium. This is not so when the resistance of this epithelial covering is lowered, as in infancy, or by trauma, or old age. However, in the urethra, cervix, and body of the uterus, the delicate cylindric epithelium does not resist gonorrheal invasion, and infection is possible, even without the loss of the surface epithelium.

The great majority of infections in the female is conveyed from chronic gonorrhea in the male. The writer believes that when a man

has once had a chronic posterior urethritis and prostatitis, he is never cured of his infection, and that under the excitation of coitus he can infect the cervix of his wife.

Nöggerath was the first to appreciate the great chronicity of the disease. Gonorrhea may exist in the genital tract during the period of pregnancy without clinical manifestations and become active during the puerperium; this explains some of the late postpartum infections. It has been proved that by passing gonococci through a second individual they may acquire added virulence; this explains how an apparently cured subject of chronic or latent gonorrhea may infect his wife, and in turn be reinfected by her. The virulence of the gonococcus is increased by menstruation, and during pregnancy, fortunately, the dried gonorrheal secretion is innocuous.

The period of inoculation with gonorrhea varies from twelve hours to a week. The part most likely to be infected depends largely on the point of contact. If the introitus is small, the urethra and Bartholin ducts are most liable to be the seat of the initial lesion; while in the *parous* woman the cervix is most often the seat of the primary infection. The infection almost invariably begins as a surface inflammation, and spreads more or less deeply into the underlying structures. The gonococcus may lie dormant in the cervical glands for a prolonged period and spring into activity under proper stimulus. On the other hand, protracted encapsulation, such as often occurs in the adnexa, tends to destroy the organisms. The gonococcus is believed to prepare the soil for subsequent infection, such as tuberculosis, or more ready invasion by the pyogenic organisms.

Under ordinary conditions the gonococcus rarely produces serious lesions in localities invested by adult squamous epithelium, the inflammatory reaction being due to the toxin ladened discharge from the lesion higher up. In the cervical and corporeal mucosa, however, a different condition exists; the surface epithelium becomes swollen, and the cells become separated from one another by the inflammatory exudate; desquamation takes place, and many of the cells are replaced by a modified epithelium, or cicatricial tissue may result; the gonococci quickly gain access to the glands and similar changes occur in the investing cells; and the exudate is poured out into the periglandular tissues, so that the gland openings become occluded and filled with inflammatory exudate, with the formation of pseudo abscesses. As the process advances, the epithelium and its basement membrane may be entirely destroyed, and a true abscess surrounded by a pyogenic membrane may form. Such is the pathology of an abscess of Bartholin's gland or of occlusion cysts in

the cervix. Gonococci in the glands may persist long after a surface cure has been effected; and from this location *reinfection* frequently occurs. This tendency to glandular penetration, which is possessed by the gonococcus, accounts for the resistance of the coccus to gonococcids applied to the mucous surfaces. From the surface and gland crypts the gonococcus escapes into the stroma and underlying muscular layer, and in severe tubal infections even into the serosa and adjacent structures. This migration excites a local tissue reaction, so that the submucous structures become swollen and infiltrated with inflammatory products and the contiguous blood vessels become intensely congested. These reactionary changes vary with the severity of the infection, the stage of the disease, and the anatomical character of the tissue involved. The underlying swelling and edema are accountable for a desquamation of the surface epithelium and distortion of the parts by the long continued inflammation in the depths of the mucosa, with the formation of cicatrices, which interfere with lymphatic drainage and consequently allow a continuance of the symptoms after all signs of active inflammation have subsided.

This is particularly evident in intraperitoneal pelvic lesions, where adhesions and contractions so distort the pelvic anatomy that operative measures for relief must be instituted for the patient's comfort and health, rather than for the cure of the infection. The most marked pathological changes are usually found near the surface, as the tissue in this locality seldom undergoes complete resolution. The chief characteristic of gonorrheal inflammation is its chronicity.

Frequency of Gonorrhea.—It seems that the time has arrived for the physician to acquaint the public with the general and widespread evil effects of gonorrhea. Neisser states that with the exception of measles, gonorrhea is the most widespread of all diseases. *"It is the most potent factor in the production of involuntary race suicide, and by sterilization and abortion does more to depopulate the country than does any other cause"* (Norris). Nöggerath said that in New York city, of 1000 married men, 800 have had gonorrhea; that 90 per cent of all these have not been cured and can infect their wives; that in New York 80 per cent of married women, at least, have gonorrhea or the results of it. While, at first glance, this seems an extravagant statement, our own observations tally very well with Nöggerath's conclusions.

In France, where statistics have been accurately compiled, it has been found that of about 10,000,000 families, 2,000,000 are without issue; these results, according to Neisser, would tend to show that gonorrhea is the etiological factor in nearly 1,000,000 sterile marriages, and this does

not include the vast number of "one child sterilities" due to this condition.

Unfortunately, owing to the secret nature of the malady, it is difficult to estimate accurately the actual frequency of the disease in civil life; for gonorrhea, attacking the genital tract below the internal os, frequently produces only mild symptoms, which are wont to be overlooked by the patient, and consequently she seeks no medical advice and has no treatment; while in chronic gonorrheal infection, the local foci are not infrequently overlooked by the average physician. Hence it will be seen that many female gonorrheics not only receive no treatment, but continue innocently spreading the infection.

In a study of 789 cases of sterility observed by the writer, over 400 proved sterile as the result of chronic pelvic inflammation, in which a history of gonorrheal infection was definitely obtainable and local lesions demonstrated. Consequently the inhibiting influence of gonorrhea upon the procreating capacity of women should have very serious consideration.

Gonorrhea of the cervix does not always prevent pregnancy, but it produces such pathology that abortion is more frequent and this in turn is more apt to be followed by a mixed infection which may extend to the tubes and peritoneum, and result in subsequent sterility.

The so-called "one child sterility" is accounted for in a large measure by the extension of a preëxisting gonorrheal infection during the puerperium, for it is a well established fact that in the puerperium the infection which was confined to the cervix and urethra is likely to extend to the uterine body and tubes, when it will almost certainly result in sterility. Besides the serious effect which the gonococcus has upon the prospective mother, it is a definite risk to the offspring, for in the passage of the child through the infected maternal parts it undergoes "a veritable baptism of virulence" (Morrow), the eyes become infected and the possibility of blindness is imminent. It is estimated that over 20 per cent of the blindness in the world may be ascribed to gonorrhea.

It is a matter of clinical observation that a woman infected with gonorrhea may transmit the infection at one time and not at another. This may be explained where the infection is confined to the urethra, for urination shortly before coitus may temporarily wash away the infective bacteria. It has also been observed that a latent infection within the uterus may not be transmitted because the secretions of the uterus do not contain gonococci, although in such cases the gonococcus can usually be found during the puerperium or immediately after the

menstrual period. It is at such times that the husband may become infected.

The more common locations of latent gonorrheal infection in married women are Bartholin's ducts, Skene's glands, and the cervical crypts; and extension frequently occurs into the uterus and pelvic peritoneum.

In infants and young maidens, the infection is usually confined to the vulva and vagina, and consequently may be spread by handling diapers, irrigation tips, etc.; or be conveyed through contact with an infected mother or nurse.

Gonorrheal Vulvitis.—Gonorrheal infection of the vulva has already been referred to under vulvitis, but there are certain peculiarities which should be emphasized.

During the acute stage the labia majora and mucosa about the clitoris and the adjacent structures are red, swollen and tender, and bathed in a profuse mucopurulent discharge, which contains gonococci and is extremely irritating to the adjacent skin surfaces. *It is peculiar to gonorrhea of the vulva that the urethra and the bartholinian glands are commonly attacked.*

Many observers claim that in finding a purulent secretion in the urethra, and on being able to express pus from Skene's tubules in the urethral floor, or in finding the mouth of the duct of Bartholin reddened, the diagnosis of a chronic gonorrheal infection is established to a moral certainty.

It is also not exceptional to find the inguinal glands enlarged and tender. But, notwithstanding the clinical value of these signs, we have always hesitated to make a positive diagnosis of acute or chronic gonococcic infection unless we have demonstrated the gonococcus in the pus. The purulent discharge often continues after the acute process has subsided and the subacute process is characterized by the same symptoms and signs, all of which however are much less pronounced. Some redness usually persists, but the swelling and edema are less, as is also the superficial sensitiveness. The discharge is yellowish or brownish in color, thick and less profuse, containing fewer gonococci than in the acute stage. This discharge, unless great care is taken as to cleanliness, forms brownish crusts on the skin surface, and when these are removed they often leave bleeding ulcers underneath.

Condyloma Acuminata are venereal warts of gonorrheal origin which result from irritation by the constant leukorrheal discharge pouring over the genitals. They are papillary outgrowths and usually appear about the vulvar vestibule, perineum or anus, occasionally on the vagina

or cervix. Multiple growths are the rule and masses of various size may be present in the same case.

Histologically the tumors are composed of a hypertrophy of the outer layers of the skin, the papillae forming the chief constituents of the growths. They are moderately well supplied with blood vessels, and because of this vascularity and the thinness of the outer layers of the epithelium they appear as whitish, pinkish or purplish wart-like cauliflower shaped masses. The surrounding connective tissue generally shows a moderate degree of chronic inflammatory reaction.

These masses may spring from a broad base, or may be distinctly pedunculated; they originate as papillary outgrowths, and may remain as such, or may coalesce. The surface of the tumors and the surrounding skin are bathed in a thin, irritating, offensive discharge.

The gonorrheal condyloma acuminata possesses a distinctly pointed apex and only when situated in the vagina become flattened as the result of pressure. When this occurs it presents a somewhat macerated appearance, the irritating discharge producing a dermatitis in the surrounding skin surfaces.

Urethritis and Bartholinitis are generally associated with gonorrheal vulvitis. Bartholinitis and bartholin abscess have already been discussed in a previous chapter among the complications of vulvitis.

Gonorrheal Urethritis.—The infection commonly originates at or just within the external urinary meatus. On inspection the mucosa of the external meatus is swollen, reddened and everted, so that the swollen mucosa protrudes from the urethra. The inflammation commonly involves the mucosa as far back as the urethrovesical junction. With the finger in the vagina, the urethra itself may be felt as a tender, more or less indurated band lying beneath the vaginal mucosa; by pressure against it with the finger we may milk it of a considerable quantity of creamy pus containing typical gonococci. As the disease becomes chronic, the discharge diminishes and becomes mucopurulent in character; the gonococci decrease and finally disappear altogether, except in Skene's tubules, which remain reddened and prominent and from which pus containing gonococci may be expressed by upward and forward pressure along the outer end of the urethra.

Gonococci may remain indefinitely in Skene's tubules, exciting from time to time a meatus inflammation and maintaining their infectivity. If the inflammation has been severe, a certain amount of peri-urethral inflammation may remain for a long time.

Abscesses may form in Skene's glands or in any of the mucous glands of the urethra. They are usually found on the floor of the anterior por-

tion of the urethra, and tend to bulge into the vagina. As the inflammation subsides, only the reddened, everted mucosa at the meatus may remain; this may involve the whole circumference, or protrude as a caruncle from the urethral floor.

Gonorrheal Vaginitis.—There is nothing characteristic in the naked eye appearance of gonorrheal vaginitis, which distinguishes it from acute vaginitis of other etiological origin. Hence, the history of exposure or marriage and the characteristic associated lesions become of extreme clinical importance in making a presumptive diagnosis, owing to the fact that after puberty the vagina is lined by a modified skin and has comparatively few glands. Acute vaginitis during sexual life is infrequent. On the other hand, in children the outer layer of the stratified squamous epithelium is ill developed, which accounts for the frequency of acute vaginitis in infants and little girls.

During the acute stage the vaginal mucosa is reddened, swollen, edematous, and bathed in a creamy, yellowish purulent discharge, which may be blood streaked. The normal acid reaction of the vaginal secretion is diminished or may be alkaline. In the acute stage the inflammation is diffuse, involving the entire vaginal tube. In the chronic stage, especially in the young and in the pregnant woman, the vaginal mucosa presents a granular appearance, due to the tendency toward localization of the various groups of vaginal papillae; consequently ulcers or small excoriations are frequently found in and about the vaginal vault.

Clinically, gonorrheal vaginitis cannot be distinguished from other forms of vaginal inflammation, without the isolation and microscopic recognition of the gonococcus.

Histologically, in the acute stage the various layers of the vaginal mucosa are swollen and infiltrated with inflammatory products, while in the chronic stage the inflammation shows a tendency to localize in certain groups of papillae in the subepithelial tissue, and the infiltration interferes with the surface blood supply; hence the epithelium may desquamate and produce numerous small ulcers, which give the sanguineous purulent character to the discharge.

On Microscopic Section, immediately beneath the stratified squamous epithelium we find a well defined zone of inflammatory reaction, i.e., an infiltration of small round cells with polymorphonuclear leukocytes; serum, injected capillaries and leukocytes are often found in this overlying epithelium.

Cervical Gonorrhea.—The vaginal portion of the cervix at the external os is a common seat of gonorrheal infection in married women, for if the introitus is relaxed, the cervix becomes the first point of con-

tact and is frequently directly infected at coitus. Gonorrheal cervicitis may also result from an extension of the vulvovaginitis in children.

According to Menge, cervix infection is found in about 80 per cent of all acute cases, and in 95 per cent of chronic cases. The gonococcus seems to have a selective affinity for the cylindric epithelium lining the cervical canal, the squamous-pavement epithelium of the portio having the same resistance as that of the vagina. The point of transition from flat to columnar epithelium, however, varies. This point of transition may be without or within the external cervical os, hence the occurrence of the infection is influenced by the location of the cylindric epithelium.

For, according to Norris, "women in whom the squamous epithelium is thin, those in whom the squamous epithelium extends only to the external os, and those in whom the so-called congenital erosions are present, should be more susceptible to gonorrheal infection than those women in whom the squamous epithelium extends deeply into the cervical canal, for gonorrheal infection tends to localize itself in the true mucosa of the cervical canal and produce an endocervicitis."

When the cervix is infected in the acute stage, there is a plentiful purulent secretion which fills the upper part of the vagina; the cervix is swollen and tender to the touch, and as a result of the hyperemia and swelling of the mucosa of the canal, the mucosa tends to evert and project itself beyond the external os, which is seen to be surrounded by a zone of congestion. If the inflammation persists, it may involve the adjacent epithelium of the portio, so that the external os appears as a bright red spot surrounded by an infiltrated granular area covered with a mucous or mucopurulent discharge. This erosion is in reality an overgrowth of the lymphoid structures within the cervical canal. It only follows that, as the cylindrical epithelium lining the canal is continuous with the cervical glands, the infection must penetrate these and thus enter the tissues of the cervix, producing a coexistent cervicitis.

In the chronic stage the cervical endometrium is rarely uniformly involved; a tenacious mucous or mucopurulent secretion bathes the surface and fills the cervical canal; the mucosa is thrown into irregular elevations, which become molded by the cervical canal into polypoid bodies. These polypi may be one or many in number, and present or protrude into the vagina; they are apt to cause a mucosanguinopurulent discharge and prolong the menstrual flow. They bleed readily and occasionally bleed on coitus. The mucosa contains numerous glands of the simple crypt and racemose type; these glands become the lurking places for the gonococcus and are the cause of the treatment being so difficult.

Histology.—During the acute stage the entire mucosa becomes edematous, infiltrated with inflammatory products, and the openings of the glands, being compressed by the inflammatory infiltration, become more contracted, so that gland drainage is impaired. As a result, some of the gland openings become blocked and retention cysts result.

Gonorrheal Endometritis.—Involvement of the corporeal endometrium is invariably an extension upward of the cervical infection. This upward invasion may occur at a menstrual period, or shortly after emptying the uterus following abortion or term delivery, or the infection may spread upward as a result of unnecessary and injudicious local treatment or instrumentation of the cervix, during cervical infection.

While gonorrheal cervicitis tends to become chronic, and exhibits little disposition toward spontaneous cure, gonorrhea of the corpus is a self limited infection, the cure being effected by resolution. This power of self defense on the part of the corporeal structures is due, first, to its rich blood supply, second, to excellent drainage from the body and the uterine contraction. Unfortunately the gonococcus, passing upward past the barrier presented by the internal os, often does not stop in its advance until the tubes have been invaded. While the corporeal endometrium almost always regenerates and becomes bacteria free (Curtis), not so with the endosalpinx; hence it is possible to conceive how the inflammation of the corporeal endometrium may become reinfected from the leaking uterine end of a pyosalpinx. In other words, chronic corporeal endometritis does not exist as a pathological entity, except in conjunction with tubal infection.

Since the work of Adler, Hitschman, Norris, Keene and Curtis, we have learned that the endometrial changes formerly described as glandular, interstitial, polypoid and fungoid endometritis, are merely phases of the menstrual cycle, often changed in their intensity by the contiguous lesions in the adnexa and adjacent peritoneum, which somewhat modify the blood supply of the uterus. For it cannot be denied that the endometrial changes during the menstrual cycle are more marked in a prolapsed, subinvoluted or retroflexed uterus, than in one in normal position with an equalized efferent and afferent circulation propelled by normal healthy uterine muscle.

Most observers consider the presence of the plasma cell a certain criterion of inflammation, and that nothing else is so positive, though Buttner concedes that an abundant infiltration of leukocytes is suggestive of inflammatory reaction.

It is now generally conceded that the diagnosis of endometritis depends on the microscopic findings. Both Frank and Norris believe that

glandular hypertrophy and hyperplasia may be due to inflammation as well as to ovarian influence. Norris states that glandular and more particularly interstitial changes, of sufficiently pronounced characteristics to warrant a diagnosis, do occur as the result of inflammation, and that, while it is nearly always possible to demonstrate the plasma cell in both acute and chronic cases, and while he considers it of great diagnostic value, still he is of the opinion it is not positively essential for the diagnosis of endometritis. Further, that the entire question should not rest on this one point; and as further proof that the glandular and interstitial changes are not entirely dependent on the menstrual cycle, he states that it is no uncommon experience to find in the same endometrium some areas presenting pronounced glandular hypertrophy, whereas in others atrophic or interstitial changes may be observed.

Gonorrheal endometritis cannot positively be distinguished from other forms of infection in this locality, except by the demonstration of the specific coccus in the tissue or in the exudate. Naturally this is possible in acute cases with their abundant purulent secretion, but very difficult in the chronic lesion with infiltration of the tissues involving part of the wall of the uterus.

In the Acute Stage the musculature is slightly increased in thickness; it is succulent from a serous infiltration, and turgid from the engorgement of the vessels, while the endometrial surface often presents a granular appearance.

Histology.—In gonorrheal endometritis the most marked changes are usually found in the superficial portions of the endometrium. The mucosa is swollen, edematous and hyperemic, and the surface may present areas of granulation; while other parts of the surface epithelium may be proliferated and more or less atypical in shape, size and qualities. The glands show various changes; some are normal, whereas others are enlarged or may be cystic. Occasionally the glands may appear to be contracted from the pressure of the inflammatory exudate in the stroma. The glandular epithelium is rarely proliferated or desquamated, but commonly shows evidence of inflammatory reaction. The stroma is infiltrated with serum, hence edematous, and contains polymorphonuclear leukocytes and blood cells. The blood vessels are congested and the myometrium is infiltrated with inflammatory products.

In the Chronic Stage the walls of the uterus are thickened, even to double their normal dimensions; there is an increase in the firmness of the texture and a loss of the normal elasticity, due to an increase in the connective tissue. This naturally disturbs the normal uterine circulation,

favoring a circulatory stasis, as the contractile power of the muscle tissue is impaired and promotes a vicious cycle.

Acute gonorrheal endometritis commonly terminates in resolution, owing to the perpendicular arrangement of the uterine cavity (when the uterus is in normal position), which favors drainage, and also to the abundant blood supply of the mucous membrane.

On the other hand, active inflammation of the endometrium is not infrequently kept up in cases of pyosalpinx by the leakage of pus through the intramural portion of the tube into the uterine cavity. Owing to the histologic arrangement of the endometrium and the investing of the myometrium with utricular glands, metritis must be regarded as a frequent accompaniment of endometritis. Furthermore, it is important to bear in mind the relative infrequency of endometritis as compared with cervicitis, and the common association of endometritis with metritis and adnexal lesions; for, unless these facts are impressed on the mind of the practitioner, the employment of intra-uterine applications and instrumentation will carry a cervical infection into the uterus with extension to the tubes, with most disastrous results. *When left to themselves, not more than ten per cent of acute gonorrheal infections of the cervix extend above the internal os.*

Symptoms of Acute Gonorrheal Endometritis.—The infection usually manifests itself shortly after a labor, abortion, or just after a menstrual period; or, the infection of the endometrium may occur during menstruation, for at these periods the open cervical canal favors the extension of the infection from below.

When the endometrium becomes involved, the inflammation may be ushered in by a chill or chilliness, which is seldom severe; fever is always present, though the temperature rarely rises above 102.5°, while both pulse and respiration are somewhat accelerated. Nausea and vomiting may occur, especially if the infection is of the severe type, as is frequently found in young married women of blonde complexion, or in women of the pituitary type; and rectal and vesical tenesmus may be present.

The woman usually complains of pain over the lower abdomen, which is most marked in the region of the uterus; while some menstrual irregularity is frequently observed. The leukorrhea, which at the onset may be diminished, becomes profuse. The discharge from the corporeal endometrium is copious and thin, unlike the thick tenacious mucus from the cervix. In gonorrhea, however, cervical infection invariably exists when the corporeal endometrium is involved, so that the discharge from the body becomes mixed with that of the cervix, giving the dis-

charge a mucopurulent, sanguinopurulent, or purulent character. It is made up of mucus, serum, epithelial débris, and pus, and contains numerous intracellular diplococci.

Should a bimanual examination be made at this time, the uterus would be found slightly enlarged, softened and tender; while the cervix, which invariably participates when the body is infected, is hypertrophied and tender, and the cervical canal is more patulous than normal.

Evidences of gonorrhea in the urethra or external genitals are nearly always present, and the gonococci may be recovered from these locations when it may be impossible to demonstrate them in the leukorrheal discharge, unless a culture is made from the interior of the uterus, which is a hazardous procedure in acute endometrial inflammation and *should never* be done.

Differential Diagnosis.—Acute gonorrheal endometritis has to be distinguished from septic endometritis of the streptococcic variety. This is done by the history and the usual absence of severe constitutional disturbance and the tendency of gonorrheal infection to become chronic. Again, gonorrheal infection frequently extends to the tubes, while septic endometritis spreads to the parametrium.

The chief points that will suggest the spread of the infection to the adnexa are (1) the persistence and severity of the symptoms, (2) pain and tenderness in the ovarian regions with overlying muscle rigidity, (3) vaginal examination showing enlarged and tender tubal masses beside or behind the uterus, (4) induration in the vaginal fornices. *Fixation and sensitiveness of the cervix always indicate extension of the disease outside of the uterus.*

Histology of Chronic Endometritis.—In the chronic stage, the pathology is usually found in the superficial position of the endometrium. The surface epithelium is flattened, and some areas may be desquamated, while in others there is active cell proliferation. The glands in these deeper portions are often enlarged and may in some instances, due to occlusion or constriction, become cystic. There is, of course, an inflammatory exudate in the stroma which disarranges the position of the glandular elements, crowding them together on the one hand, or widely separating them on the other. The glandular epithelium undergoes similar changes to that of the surface, though these changes are, as a rule, less pronounced; consequently the gland crypt may be empty or contain serum, leukocytes, blood and epithelial débris, while the periglandular stroma is densely infiltrated with inflammatory products.

The blood vessels, which normally consist only of endothelial tubes, often possess well developed muscular walls and are increased in number.

These superficial changes, which extend into the basal membrane, necessarily involve the underlying uterine muscle in the inflammatory process and produce varying degrees of metritis.

Pathology and Histology of Gonorrheal Metritis.—Gonorrheal inflammation of the uterus may be limited to the cervical and corporeal mucosa, but usually involves the underlying myometrium to a greater or less extent, owing to the peculiar placement of the utricular glands and the uterine lymphatics. In severe cases the inner layer of the uterine parenchyma is always invaded and infiltrated with inflammatory products. This causes the uterus to become enlarged, softened and boggy; the enlargement is general, but especially in the transverse diameters. Pronounced cases of gonorrheal metritis are commonly associated with adnexal and peritoneal inflammations, which help to keep up the circulatory stasis with the subsequent tissue changes.

Symptoms of Chronic Gonorrheal Endometritis.—Chronic gonorrheal endometritis may occur as a sequela of an acute infection, or originate as a subacute process. When it does occur, it is always associated with gonorrhea of the cervix; hence it will be seen why acute exacerbations frequently follow intra-uterine applications or instrumentation, particularly when these manipulations occur near a menstrual period or after a miscarriage.

The most constant symptom of chronic gonorrheal endometritis and endocervicitis is leukorrhea. The discharge is usually whitish or yellowish in color and thinner than that originating in the cervix. Microscopically the secretion is composed of serum, epithelial débris, leukocytes and a few red corpuscles.

Most authorities (Hitschman, Adler, Keene, Norris and others) claim that no mucus is secreted from the glands of the endometrium except near the menstrual period, hence any mucus found in the leukorrheal discharge must necessarily be of cervical origin.

In chronic endometritis it is extremely difficult to recover the gonococcus from the discharge, for not only are the germs few in number, but they have a habit of lying dormant in the deeper tissues and appear only during exacerbations.

Menstrual disturbances, as amenorrhea, menorrhagia, and metrorrhagia, are not infrequent, though profuse menstruation is the most frequent anomaly. Congestive dysmenorrhea is a common symptom; the pain usually persists during the first few days of the flow. As stated by Norris, dysmenorrhea, occurring in women in whom menstruation has previously been painless, and in the absence of other gross lesions, especially if gonorrhea of other parts of the genital tract exists, becomes

a most suggestive symptom. It is also generally admitted that gonorrheal endometritis is a frequent cause of sterility, though in our experience, gonorrheal cervicitis is really one of the most common causes and not the inflammation of the corporeal endometrium.

Finally, there is usually found some degree of enlargement of the uterus, due to the associated metritis. Knowing the power of the endometrium to sterilize itself of bacteria when the uterus is in normal position and proper drainage is established, it seems strange that so much attention has been given to chronic gonorrheal endometritis. On the other hand, the cervix is never able to rid itself of an infection, hence we have come to believe that the chronicity of corporeal infection never exists clinically, unless it is associated with inflammation of the tubes.

Gonorrheal Inflammation of the Fallopian Tubes.—Tubal inflammation of gonorrheal origin is an extension of the infection from the uterine endometrium by direct continuity along the mucosa. The disease begins as an endosalpingitis, but the infection rapidly invades the muscular and serous coats and excites a contiguous peritonitis.

While the disease is always bilateral, a different degree of pathology may exist in each tube; as, for example, one tube may be the seat of a suppurative salpingitis, while in the other the infection may have terminated in a pyosalpinx. Generally speaking, the more frequent, prolonged, and severe the attacks of pelvic peritonitis have been, the more extensive the tubal pathology is likely to be. The most frequent pathologic lesion produced by a gonorrheal infection of the tube is a pyosalpinx.

Certain Characteristics Are Peculiar to Gonorrheal Infection.—In gonorrhea the tubal mucosa is primarily invaded, while streptococci and staphylococci reach the tubes, either by the blood or lymphatic channels of the broad ligaments; hence, in these forms of infection, parametritis is a constant associated lesion and perisalpingitis the result. *This parametrial involvement is absent in pure gonococcic infection* and, if cellulitis is present, it is usually secondary to salpingitis, being due to the pouring out of exudate into and through the tubal wall.

In tubal tuberculosis, which makes up about eight per cent of all tubal inflammations, the lesion is frequently secondary to tuberculosis in other parts of the body. It is easily recognized at the operating table, as it produces a distinctive pathology. Small miliary tubercles scattered over the surface of the tube, the imperfect closure of the abdominal ostium with a few fimbriae protruding from the partially closed

ostium, and the cheesy contents are macroscopic proof of the nature of the infection.

An absolute diagnosis of tuberculosis can almost always be made with the aid of the microscope. On the other hand, tubal infections secondary to pelvic peritonitis, due to postpartum lesions or appendicitis, affect the outer coats, producing a perisalpingitis, while the endosalpinx is more or less normal in appearance.

Definite Microscopic Appearances Are Noted in Gonorrheal Salpingitis.—During the acute stage the tubes become elongated and swollen, kinked and bent upon themselves. The surface is markedly congested, owing to the increased vascularity, and adhesions are nearly always present. The increased weight of the tube usually carries it down into the *cul de sac;* consequently the distal or free portion of the tube shows the greatest disturbance, since this portion is nearer the abdominal ostium, through which the infective material must escape. Hence the greatest reaction is excited in the adjacent peritoneum, where a plastic exudate is poured out.

On section, the walls of the tube are found to be soft, congested, and edematous, while the mucous folds are reddened, swollen, and bathed in a purulent or seropurulent exudate. As the disease tends to become chronic, the abdominal ostium closes, either by becoming attached to some adjacent viscus, or to the parietal peritoneum in the *cul de sac,* or by inversion of the fimbriae and adhesion of their peritoneal surfaces. When closure occurs by adhesion to some contiguous organ or the pelvic peritoneum, the closure is *seldom complete, and intermittent leakage of the infective contents is apt to occur,* which explains the frequent exacerbations of peritoneal inflammation so common in subacute and chronic gonorrheal tubal inflammation.

If the disease tends to become chronic without closure of the external abdominal ostium, the adhesions on the surface become more dense and less vascular and the walls become moderately firm, due to the increase in fibrous connective tissue.

Histology.—At first the inflammatory reaction is confined to the mucosa, later there is always more or less involvement of the muscularis; as the various coats become infiltrated with inflammatory products the surface epithelium presents evidences of inflammation, but it is rarely desquamated or proliferated, the inflammatory products tending to extend through the muscularis along the lymph or blood vessels, where groups of small round cells or polymorphonuclear leukcocytes may be found.

Pyosalpinx.—A pyosalpinx is the usual termination of a gonorrheal

salpingitis. This results from the closure of the abdominal ostium by inversion and agglutination of the peritoneal surfaces of the fimbriae. How this is actually accomplished has been the subject of much discussion. Whether it is due to an increase in the total length of the tube wall, which, by expanding in an outward direction, becomes projected beyond the tubal fimbriae, and thus allows the agglutination; or whether, as suggested by Ries, there is a gliding outward of the "peritoneal ring" over the fimbriae, which is made possible by the fact that the walls become loose and redundant, subsequent to the collapse of the distended tube, seems of little consequence, so long as inversion and adhesion of the fimbriae by their peritoneal surfaces actually occurs, for when this takes place the retained contents are sealed and further infection from leakage is prevented.

On the other hand, the perimetric or peritonitic closure described by Doran, which consists of a matting together of the fimbriae by inflammatory adhesions without preliminary recession, seldom completely closes the ostium and infective leakage frequently occurs.

These purulent accumulations form a sausage shaped tumor and are chiefly found in the outer two thirds of the tube; while the isthmic and interstitial portions show some thickening, but little enlargement. These pyosalpinges vary markedly in size, from large retort shaped swellings, with thin friable walls, filling the entire pelvis and extending up into the abdominal cavity, to small pyosalpinges with immensely thickened coats.

In recent cases the gonococcus can be recovered from the pus by either culture or smear, but in old cases of long standing the pus is usually sterile. In recent cases the mucosa is commonly found intact, while in chronic cases of long standing the mucosa is entirely disintegrated and replaced with a pyogenic membrane or by granulation tissue.

Clinical Course of Gonorrheal Inflammation of the Tubes and Ovaries.—Gonorrheal inflammation of the endometrium frequently extends to the tubes and from the tubes to the ovaries and pelvic peritoneum. This complete invasion has been observed to take place in less than two weeks from the time of the initial infection of the cervix. The rapidity of the advance, however, is dependent on the virulence of the infection and the resistance of the tissues. When the infection occurs near the menstrual period, or at the time of emptying a gravid uterus, extension to the tubes and pelvic peritoneum is very rapid.

Our clinical experience would seem to indicate that when the corporeal endometrium is infected, the disease extends to the tubes and from these to the ovaries. Nevertheless it has been impossible to establish

with any accuracy the frequency of the invasion of the adnexa. Menge, in combining the statistics of Bumm, Sternschneider, Fabry, Brüschke, Bröse and Welander, found that the tubes, ovaries and pelvic peritoneum were involved in 25 per cent of acute, and 50 per cent of the chronic cases.

As we have already stated, the primary tubal lesion is an *endosal-*

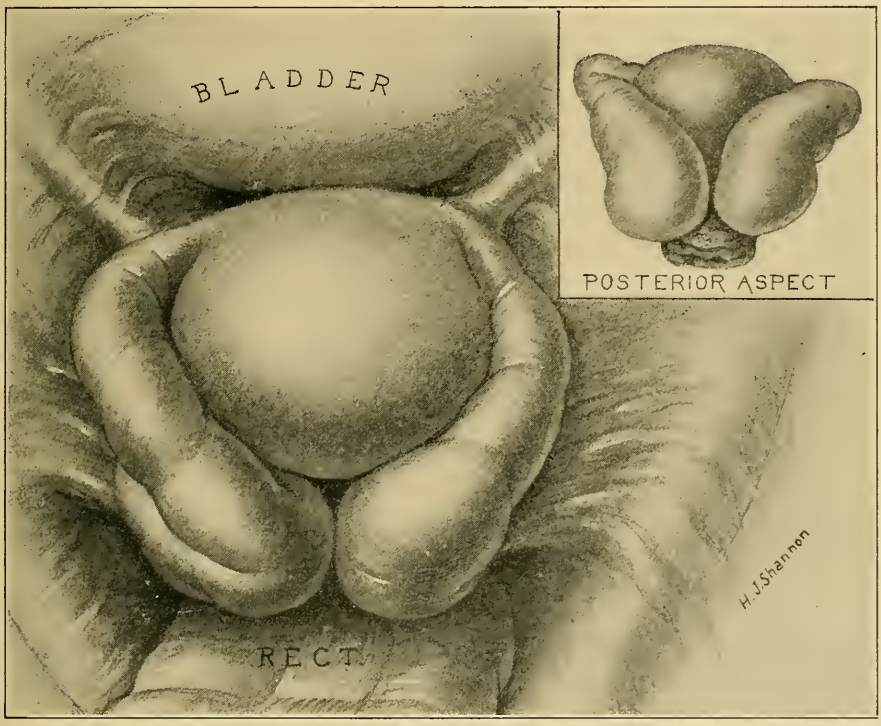

FIG. 20.—PHOTOGRAPH FROM AUTHOR'S COLLECTION, SHOWING HOW A DOUBLE PYOSALPINX ROLLS OVER AND COVERS THE OVARY. BOTH TUBES ARE ADHERENT IN THE CUL DE SAC.

pingitis, but the inflammation quickly spreads to the deeper layers of the tubes, involving the muscularis and serosa.

Gonococci have repeatedly been found in the depths of the tubal wall; *hence there must be an accompanying cellulitis in the broad ligament in extensive tubal lesions.* As a result of the salpingitis, an inflammatory exudate forms, which escapes through the tubal wall, the abdominal ostium and produces a peri-oöphoritis and pelvic peritonitis. Fortunately the tunica of the ovary offers a substantial resistance to infection from without, and unless the infection directly invades a ruptured follicle, *abscess of the ovary is unlikely from gonorrheal origin.* On the other hand, local peritonitis is common, and the adjacent pelvic structures be-

come matted together with the adnexa by extensive adhesions. During the exacerbations of tubal inflammation, which may be excited by coitus, intra-uterine instrumentation or medication, more exudate escapes from the tubal openings or through the walls of the distended tube, and further peritoneal reaction is excited, owing to the increased weight of the free portion of the tube, which is always the site of the greatest inflammatory reaction; it rolls over and covers the ovary, and both become prolapsed and adherent as tender swollen masses in the *cul de sac* of Douglas, small purulent accumulations being not infrequently found between the adnexa and the adjacent structures, more or less completely walled off from the general peritoneal cavity by the sigmoid and omentum. It is not unusual to find on one side a large inflammatory mass composed of a pyosalpinx and an inflamed ovary, while the other tube, though infected, may have incurred little or no damage, except from the associated perisalpingitis. When there have been repeated attacks of pelvic peritonitis, it is exceptional to find normal structures on either side.

In the study of the individual case, it must be remembered that pelvic inflammatory disease may be produced by other germs than the gonococcus alone and in combination; that the gonococcus prepares the soil for other forms of infection, must also be admitted. Hence the etiology of each case of pelvic inflammation becomes of the utmost importance, for both the prognosis and treatment are largely dependent upon the type of infection.

While it is not always possible for the surgeon to know positively the form of infection he is dealing with, clinical experience has taught us that an accurate history and appreciation of the site of the local lesions makes a presumptive diagnosis possible.

Generally speaking, the majority of pelvic infections may be grouped under three headings, gonococcal, pyogenic, and tuberculous, and their incidence has been variously estimated by different observers.

Norris, in his "Gonorrhea in Women," combines the statistics of Andrews, Menge, Krönig, Miller, Hyde, Pankow and Wertheim, and shows that in specimens removed from the 3501 cases studied the gonococcus was demonstrated in lesions 178 times or in 17.4 per cent of the total. Such a low incidence, however, does not represent the actual proportion of cases which were of gonorrheal origin; for operation in the acute stage is practically never done, and in chronic cases, where there has been long encapsulation of the infecting organism, the gonococcus is seldom found, for the organism has been destroyed either by its own toxins or the migration of the colon bacillus.

We would feel from our clinical experience that over 75 per cent of all pelvic infections have a gonorrheal origin. The importance of accurate diagnosis, as to the type of infecting organism, is shown by a study of the behavior of the various organisms within the tubes. A large proportion of gonorrheal tubes ultimately become sterile after a period of from six weeks to three months; the death of the infecting gonococcus can be assured if both ends of the tube are closed; it lives longer if leakage occurs. On the other hand, the pyogenic bacteria have an unknown longevity, even when encapsulated, and retain their virulence for extremely long periods.

Symptoms of Acute Pelvic Inflammation.—As an acute gonorrheal salpingitis cannot occur as a distinct pathological entity without associated infection of the adjacent viscera and peritoneum, it is impossible to have a symptomatology that does not include the symptoms and signs of the peritoneal reaction.

Consequently the symptoms must vary with the extent of the lesion and the stage of the disease. The extension of the infection from the uterus to the fallopian tube usually follows a menstrual period or the emptying of a gravid uterus; or, when the primary infection is in the cervical tissues, upward extension is usually spread by some form of intra-uterine instrumentation, such as a curetting or the introduction of a stem pessary.

The subjective symptoms are fever, pelvic pain, leukorrhea, and a bloody vaginal discharge. The pain and tenderness are diffused over the lower abdomen, usually more marked on one side than on the other. Pain may be considered as a diagnostic feature, for it is always more severe when the inflammation extends to the tubes than when it is confined to the uterus. It is usually intermenstrual during the acute stage. Owing to the fact that the tubal lesion is only a part of the general pelvic inflammation, the exact symptomatology is difficult. Usually when the infection involves the tubes there is a chill, followed by nausea, malaise, headache, and elevation of temperature, and an increase in the pulse rate. In cases of gonorrheal origin the temperature rarely rises above 103.5°F. or the pulse rate above 120. Commonly the readings are below these figures; the blood count shows a leukocytosis and owing to the tendency of the inflamed tube to fall into the *cul de sac* of Douglas, rectal tenesmus is frequently complained of.

As we have already stated in the discussion of the pathology, the disease may be unilateral or bilateral, the tubes being involved simultaneously, or one tube may be attacked at a time. In severe cases the wide spread abdominal tenderness, pain, and tympanites give a clinical picture

of general peritonitis, and not until some of these acute symptoms have subsided is it possible to recognize by vaginal or abdominal examination the actual evidences of tubal involvement. Inspection will usually reveal evidences of gonorrhea in the lower genital tract at one or more of the common locations, i.e., at the meatus, in Skene's glands, or the mouths of Bartholin's ducts. The cervix is enlarged, soft and tender, always more or less fixed, and very sensitive to motion; on inspection, it is congested and a purulent or mucopurulent discharge may be seen issuing from the external os. The uterus is enlarged, softened and tender, and induration and sensitiveness are present in one or both fornices. As the severity of the acute symptoms subside, it may be possible to recognize on bimanual palpation an inflammatory mass of varying size occupying the region of the appendages during the acute stage. However, owing to the coincident peritoneal reaction with its tension, tenderness, distention, and peritoneal exudate, it is often impossible to outline adnexal lesions. It takes from a week to three weeks for the acute symptoms to subside and make detailed palpation possible.

Prognosis.—Tubal involvement by the gonococcus always leaves a definite pathology, though complete regeneration of the tube is possible, provided the infection does not become a mixed one and pyogenic cocci do not complicate the original lesion.

Fortunately, unless other bacteria participate in the general inflammation, the disease rarely if ever ends fatally, but usually subsides and becomes subacute or chronic, only to relight at irregular intervals.

Chronic Pelvic Inflammation.—In chronic pelvic inflammation there is usually a history of an acute primary infection with gradual subsidence of symptoms, although some cases seem to have followed a chronic course from the very beginning. Acute exacerbations of the disease occur at irregular intervals, due to periodical leakage of the tubal contents, and while these exacerbations may occur at any time, they are more prone to occur at a menstrual period, after emptying the gravid uterus, during the puerperium, or after intra-uterine instrumentation, or excessive sexual relations.

The woman with chronic pelvic inflammation is always more or less invalided, owing to the inflammation in and about the adjacent structures. Menstrual disturbances are common; these manifest themselves in menorrhagia, metrorrhagia, and dysmenorrhea. *There are always present symptoms of cervical infection;* in some cases amenorrhea or scanty menstruation may be noted. This is more often found where there have been repeated severe attacks of pelvic peritonitis, for in such cases the peri-oöphoritis produced thickens the ovarian tunic and interferes

with normal ovulation. It is difficult to conceive how an ovary can remain in such company without participating in the inflammatory changes.

In a few cases of hydrosalpinx, intermittent discharge of its content may occur through the uterus, and this may be followed by temporary relief of the pelvic symptoms and a subsidence of the tubal mass.

Pain in the lower abdomen referable to the site or sites of the lesions is commonly present, and not infrequently, because of the incarceration of the tubal mass, pressure pains are complained of in the thighs and external genitals.

There are always digestive disturbances, due to the intestinal adhesions which limit the peristaltic movement of the intestines. These patients complain of gas, distention, and intestinal colic. On the other hand, in time the inflammatory process may become quiescent, and the patient regain her physical tone. However, after complete subsidence pain may usually be excited by the use of cathartics and, in turn, be relieved by rest, heat to the abdomen, and enemata. Where the tubes and ovaries are prolapsed and adherent in the *cul de sac,* defecation is always painful and constipation becomes a marked feature. Fecal stasis increases the pelvic congestion, and this in time adds to the pelvic pain and backache.

Treatment of Gonorrheal Infection of the Female Genitals. —While specific infection in its acute stage differs but little from other pyogenic inflammations, the peculiar characteristics of the gonococcus, seeking, as it does, the glandular recesses, make its management and cure more difficult.

In considering the treatment during the acute stage of gonorrheal infection, *two general principles must be recognized.* (1) When the diagnosis of acute gonorrheal infection has once been established, every effort should be made to prevent its upward extension along the mucous surfaces of the genital organs.

(2) *We must attempt to completely eradicate the infection* by destruction of the gonococcus in the involved areas. Besides this, it will be necessary to relieve the patient of the burning and suffering occasioned by the profuse, purulent discharge which is always characteristic of this form of infection; and furthermore, we must instruct her as to the dangers and the possibility of contamination of herself and of others by the discharge.

In children the infection is commonly more or less completely confined to the vulvovaginal orifice, as the intact hymen virtually acts as a barrier to the upward progress of the disease; but when it once passes into the vagina, the inflammatory reaction becomes intense and, as a rule.

the vulva, vagina and cervix are all involved, and occasionally the uterus and tubes become invaded.

In the non-pregnant adult, the mucosa of the vagina is particularly resistant to the gonococcus, and therefore acts as a natural barrier to the infection. On the other hand, the *vagina of the pregnant woman is less resistant,* and consequently often becomes the seat of gonorrheal infection, which, in turn, owing to the open cervix and lochial discharge, invades the uterus and tubes during the puerperium.

The physician attending a woman during the acute stage of a vulvovaginal infection should be impressed with the importance of not carrying the gonococcus higher up by examinations and by various attempts at local treatment through an infected introitus; and, before determining what form of local treatment shall be instituted, the external genitalia should be inspected for any inflammations, reddened areas, ulcers, or papillomata. The amount and character of the discharge should be determined, and on separation of the labia, the condition of the hymen observed. This exposure will also permit inspection of the openings of Bartholin's ducts.

Inflammation of any type involving either the gland or its duct causes a reddening about the duct opening. The typical maculae gonorrheae of Sänger, which are considered as more or less pathognomonic, appear as reddened, elevated areas of three to five millimeters in diameter, having the appearance of a flea bite. Many times, however, *the only evidence of infection is a slight reddening about the orifice of the duct.* Cultures should be made from the expressed secretion.

The urethra is next examined; when it is infected there is reddening of the urinary meatus; the external orifice should be wiped dry with sterile cotton, and the urethra milked by pressure with the finger from *behind forward;* this exposes the orifices of Skene's tubules, which appear as small reddened pits, from which a drop of pus exudes.

The cervix is the next point of inspection; to do this, great care must be used *not to carry the infection up from the vulvovaginal orifice by our manipulations.* The labia are separated and wiped dry with sterile cotton, and then sponged with a 1-1000 bichlorid solution. When all discharge about the introitus has been removed, the cervix is exposed through a suitable speculum—a small trivalve or a Ferguson tubular speculum.

An intensely reddened area surrounding the external os, with pus or mucopus exuding from the os, is always characteristic of specific infection, *though the positive diagnosis in the acute stage must never be made without bacteriological proof.* When the extent of the infection

has been determined by such a routine inspection, the inflammation should be confined as nearly as possible to the area or areas involved. This may be done by (1), *physical rest* and (2) *local cleanliness. Cleanliness* is the watchword in the treatment, for the profuseness of the discharge is not only very annoying to the patient, because of its irritating effect on the mucous membranes and the adjacent surfaces, but offers considerable danger from contamination of the patient herself and those taking care of her. The bacteria and their toxins are very irritating, and result in erythema or dermatitis about the vulvovaginal orifice. When the patient suffering from acute gonorrhea presents herself for treatment, and the above investigations have been made, *our first instruction should be rest, and this rest must be in bed.* The hair about the external genitals should be removed with scissors, for this allows more thorough cleansing of the infected region and thus aids materially in the subsequent care. Coupled with this the woman should be advised—

(1) To drink copiously of water to increase her kidney function, and so, by increasing the urinary output, wash away the purulent secretions from the urethra.

(2) The diet should be restricted; all meat, spices and condiments excluded.

(3) In addition to this, *postural drainage of the genital tract should be employed;* this may be done by *using the elevated trunk posture of Fowler,* and from time to time having the patient change her position by allowing her to lie on the abdomen, in order that the discharge in the upper part of the vagina may escape from the vulva.

(4) Repeated vulvar irrigations should be made with the patient on a douche pan, the labia separated, and the irrigant poured from a pitcher over the vulvovaginal orifice. It matters not what irrigant is used, as the vulvovaginal cleansing is wholly mechanical. However, our best results have been obtained by using bland alkaline irrigants, such as a combination of bicarbonate of soda and borax, in proportions of a tablespoonful of each to two quarts of hot water, after the pus has been washed away by the solvent.

(5) *Antigonococcic solutions may be used.* Of these, bichlorid of mercury 1 :8000, phenol 1 :40, or permanganate of potassium or protargol solution all have a place. In our experience, permanganate of potassium has stood the test better than most of the other antiseptics, after the vulvovaginal orifice has been thoroughly cleansed and dried with sterile cotton.

(6) The vulva is wiped with cotton pledgets soaked in a 25 per cent argyrol or 2 per cent silver nitrate solution. Routine vaginal douches

are not employed, for, no matter how carefully the vaginal douche is given, it is bound to disturb local conditions, break down the immunity, and spread the infection upward. Even when the vagina itself is involved, *the vaginal douche is seldom indicated in the acute stage of gonorrhea,* for postural drainage and local cleanliness secure the removal of the purulent discharge; and when it is necessary, because of the severity of the inflammation, to treat the vaginal mucosa, we have found it better to make direct applications of silver solution to the vaginal walls, for in our practice we make every effort to avoid the use of vaginal irrigations until all of the acute symptoms have subsided, when we make direct applications of silver preparations to the infected areas at the meatus, in the urethra, in the vagina and cervix. This may be done after carefully cleansing the introitus by introducing a skeleton trivalve and loosely packing the vagina with gauze soaked in a 10 per cent solution of protargol; a piece of cotton wet with the same solution may be placed between the labia. Should the vulvar pain be considerable, sterile gauze wet with aluminum acetate solution, over which an ice bag is applied, will give the patient immediate comfort.

From long experience in the study and management of infection, to us it seems better to *allow nature to establish its immunity, which is the basic principle in every infective inflammation.* It is true that this plan of treatment is less radical, and oftentimes takes longer, but the complications are fewer than when more active measures are employed. We can limit extension by *favoring drainage with proper posture and physical rest, until the acute symptoms have subsided,* when an attempt should be made to *eradicate the gonococcus from the area that has been involved.*

This may be done by gonococcids which are easily applied to the surface of the vagina and the vaginal surfaces of the cervix; but when the gonococcus has invaded the tubular glands of the urethra, Bartholin's ducts, or the glands of the cervix, its eradication by any method except the total destruction of the tissue involved, offers little chance of permanent cure.

After each defecation or urination, the external genitalia should be carefully cleansed with a weak antiseptic solution. *Rectal examinations, the administration of enemata, or the introduction of suppositories must be absolutely prohibited during the acute stage, with its profuse purulent discharge,* for infection of the rectum is most intractable.

The favorite time for extension of a cervix gonorrhea to the endometrium and tubes is during the last two days of menstruation, and during the first few days following. Consequently it is advisable that the

patient should be at rest at these periods, and that all active treatment be temporarily suspended.

Individual immunity is something that is variable, for some women seem to be so resistant to acute specific infection that they fail to have any noticable manifestations of the acute stage.

Treatment of Acute Specific Urethritis.—Acute urethral inflammation causes the patient to suffer from many distressing symptoms, such as painful and frequent urination. The *most severe pain usually occurs during the act of micturition,* and then subsides slowly. To control this, the urine must be rendered bland and non-irritant by the exclusion of acids, condiments, and stimulants, the strict adherence to a milk diet, and the administration of copious quantities of pure or alkaline water. The relief thus given is further enhanced by the administration of benzoate of soda or benzoate of ammonium in ten grain doses every three hours in a glass of water. Salol in five grain doses every two or three hours in warm water may be employed instead, and rapidly renders the urine bland and non-irritating. Unfortunately the continued use of all of these drugs is apt to upset the stomach. *Not until all of the acute symptoms have subsided should any direct application or injection be made* to the urethra. For the capacity of the urethra is about fifteen or twenty drops; hence, an irrigation of greater quantity or an instillation of a larger amount will force the solution into the bladder and so extend the infection past the vesical neck and open the way for an ascending infection of the urinary tract. For this reason we are opposed to having instillations used or urethral irrigations made, but when the inflammation has become subacute, prefer to make direct application of solutions of argyrol, 25 per cent, or silver nitrate, two to four per cent, through an endoscope to the infected and inflamed areas within the urethra. These rapidly relieve the pain and diminish the hyperemia. After the inflammation has become subacute it will be noticed that the urethral infection is not general, but is confined to congested patches at various points in the canal. These should have direct applications of a two or four per cent solution of nitrate of silver at intervals of two or four days, to eradicate the gonococcus, while internally we administer some preparation of sandalwood oil.

By following out such a program, the relief of the pain on urination is usually pronounced. Occasionally, however, the pain and tenesmus may be so severe and intractable that it may be necessary to administer an anodyne. Codein in generous quantities serves this purpose and, after the acute discharge has ceased, may be employed in the form of codein suppositories.

One general principle should never be forgotten in the treatment of urethral inflammation. That is, that *too active treatment increases the inflammation and is distressing to the patient.*

The fundamental principle is to render the urine bland, and allow the disease to become confined to certain areas within the urethra, when it may be eradicated by direct destruction of the infection in the diseased areas.

The vesical neck, like the internal cervical os, is a natural barrier to upward extension, and, unless the treatment is so conducted as to cause the infection to pass along through the vesical neck, it will remain in the urethra and in Skene's tubules and not involve the bladder.

In the subacute and chronic stage Skene's tubules become the principal seat of the latent infection, retaining the gonococcus for weeks and months after it has disappeared from the urethra. These, not infrequently, cause repeated reinfections of the lower portion of the urethra; but the disease in these tubules may be completely eradicated by their destruction. This can be done after first anesthetizing the ducts with cocain or novocain, by injection into the tubules with the probe pointed hypodermic needle of one or two drops of pure carbolic acid, or by passing into the tubule a very fine cautery knife, cold, and when in place, turning on the electric current; this destroys the tubule by opening it up into the urethra. Only in this way are the tubules completely destroyed and the latent infection in them eradicated.

Treatment of Acute Specific Vaginitis.—The management of acute specific vaginitis differs in no wise from the care of acute vaginitis of other origins, except in calling for care to prevent the contamination of the patient and others by dissemination of the infective discharge.

The patient should be kept absolutely quiet in bed; the diet must be confined to non-irritating articles of food. The free exhibition of saline laxatives is advisable, and the patient should be encouraged to drink large quantities of alkaline waters. Here again, rest and cleanliness are the two fundamental principles in the management of the acute stage. Repeated vulvar irrigations of warm boric acid may be used, and if the purulent discharge is very profuse, a vaginal douche may be given. The bag should be at a very low elevation, the nozzle introduced just within the introitus, and the *solution allowed to run in and run out under low pressure.* The ordinary vaginal douche, as given by the untrained nurse or physician, does more harm than good in acute vaginal inflammation. Therefore it is our practice to abolish as much as possible the routine use of vaginal douches, and allow the patient to develop her immunity against the infective bacteria.

When, however, the acute symptoms have subsided, direct antigonococcal applications to the vaginal walls, vaginal vault, and portial cervix may be made, followed by carefully drying the walls and other surfaces with sterile cotton and their separation maintained by the introduction of gauze tampons saturated with picric acid and glycerin or saturated with a protargol or argyrol solution. These tampons should remain in place for from twelve to twenty-four hours, and then be removed; when the surface of the vagina is again exposed through a skeleton trivalve, and swabbed with a 25 per cent argyrol solution or a 10 per cent protargol solution. Time should be taken for this application to thoroughly dry, when a powder of bismuth or bismuth and starch, or of warm Fuller's earth may be blown into the vagina over all of the vaginal surfaces.

This plan of treatment rapidly controls the discharge. Only after the acute and subacute stage has passed are routine douches permissible; then permanganate of potassium of a strength sufficient to make the water a port wine color may be used as a douche two or three times daily, with the patient in the recumbent position on a douche pan, the bag at a low elevation, and the nozzle introduced just within the vulvovaginal introitus.

The external cervix, its portio, and the external os are almost always involved when there is a specific vaginitis. Here again, the treatment should be direct. The cervix should be exposed with a proper speculum, and all of its surfaces swabbed with a solution of ten to twenty grains to the ounce of silver nitrate, which must always be allowed to dry after it has been applied. This is then followed by filling the vaginal fornices, surrounding the cervix, and covering the exposed portions of the cervix with warm Fuller's earth, which is held in position with a loose gauze tampon; the gauze is removed in about twelve hours, when a saline or alkaline douche is given and the surfaces cleansed. This plan of treatment will control the infection, except that portion of it which has entered the cervical canal and found its way into the glands of the cervix.

When it has reached this point, local treatment has little avail, and the infection always becomes chronic, causing considerable cervical hyperplasia, gland proliferation, and parametrial extension into the cellular structures at the base of the broad ligaments and into the connective tissues in the uterosacrals.

If our treatment *has not been too radical, the infection will remain on the surface of the cervix, and in the cervical canal below the internal os,* and gradually localize itself in the cervical tissues, where it becomes chronic, causing a cystic cervicitis.

While many different methods of treatment have been suggested for

the management of chronic, cystic cervicitis, with its intractable leukorrhea and menstrual anomalies, but two have our endorsement. Either the entire glandular area of the cervix must be excised, as suggested by Sturmdorf, by employing the method which is illustrated under the treatment of chronic cervicitis, or else the gland structures must be destroyed by the substitution of scar tissue, as can be done by the use of radium within the cervical canal. The latter method, so far, has not been employed long enough to know its effect on subsequent pregnancies, or how the cervical tissues will act during dilatation in subsequent parturitions; while, on the other hand, excision of the cervix has been used successfully in the cure of sterility and offers no obstruction to cervical dilatation.

Acute gonorrheal endometritis is managed on the same general principles that acute infections of the uterus are treated on when the inflammation is due to other infecting agents, namely, *by rest, posture, ice bags, and anodynes.*

All acute inflammations of the uterus require (1) rest in bed in the elevated trunk posture of Fowler, to secure postural drainage; (2) the application of an ice bag above the tubes for the relief of the uterine pain and the control of infectious extension to the parametrium; (3) the use of moderate amounts of anodynes for the relief of the uterine and pelvic pain, and (4) the administration of saline to relieve the pelvic congestion.

The uterus is capable, as has been shown by Curtis, Norris, Keen, and others, of ridding itself of bacteria by its own bactericidal powers. Clinically, we have shown that it is common for acute inflammation within the uterus to end spontaneously under this general plan of treatment.

No local applications, no irrigation, no instrumentation is indicated in the acute stage of any gonorrheal extension to the cavity of the uterus.

Unfortunately, extension to the endometrium means extension to the tubal mucosa, and here again no local treatment will have any effect on the actual pathological changes which take place within the tubes.

Only rest and time will have any effect. The patient will establish her own immunity and, as best she can, resist the extension of the infective invasion.

The treatment of the results of these infections, namely, that of pus tubes, pelvic peritonitis, and their complications, will be thoroughly discussed under the heading of "Salpingitis," and it is not necessary in this chapter to make any reference to the detailed management, except to say that the general plan of treatment is rest, posture, and enemata

to relieve congestion of the lower bowel, and finally sufficient anodyne to make the patient comfortable.

These principles, with time, will result in a localized infection in the tubes or pelvic peritoneum.

When all of the acute symptoms have subsided, and the temperature has remained normal for at least a week, the leukocyte count has remained under 11,000, the exudate has become insensitive and is not painful on manipulation, and the temperature is not elevated as a result of an examination, then, and not until then, are any surgical procedures justifiable.

Long experience has taught us that the less active the treatment the better the result, and the less pathology will be found in the pelvis when the time comes for operative interference.

Finally, the question is often asked, How can we determine that a gonorrhea has been cured? To determine this, our examinations must be thorough and repeated; at least three repeated negative examinations conducted over a considerable period should show (1) the absence of the gonococcus, (2) the absence of pyogenic bacteria, (3) the absence of pus cells in the locations most commonly affected, namely the urethra, Skene's glands, Bartholin's ducts, and the cervical glands.

CHAPTER III

PELVIC INFECTIONS

Pelvic infections—Bacteriology—Mode of invasion.

Acute puerperal and non-puerperal infections of the female genital organs and their sequelae make up the largest group of the diseases peculiar to women, with which the obstetric surgeon and gynecologist has to deal.

Infection of the female genitalia takes place either from without, through known avenues of entry, or from within by a hematogenous route, in which case the original focus may be remote from the pelvis. The peculiar anatomic arrangement of the generative organs in women, constantly exposed as they are to infection and trauma, directly favors inflammatory changes. Furthermore, certain periods in a woman's life tend to subject her to infection of different types and in different locations; thus we find in infancy, with the vaginal introitus protected by an intact hymen, that infections of the vulva and introitus are most common. During puberty and adolescence, hematogenous infections of the endometrium, tubes, and ovaries, occur as local manifestations of the exanthemata and of tuberculosis, and should receive consideration. While, during the period of sexual maturity, with marriage and child bearing, the woman may be subject to all forms of gonorrheal and septic infection.

Bacteriology.—Pelvic infections, as do infections of other tissues, result from the introduction and propagation of infective organisms into a favorable soil. In general we have two classes of infective bacteria, the cocci, and the bacilli. In the former class are found the gonococci, streptococci, staphylococci, pneumococci, etc.; and in the latter may be mentioned the bacillus coli communis, and the bacillus aërogenes capsulatus. These are the common germs which play a part in the causation of acute pelvic infection, and have a practical significance from both the clinical and bacteriological standpoints.

The gonococcus of Neisser is preëminently a surface germ, invading the genital tract by continuity of structure, and negativing the acid secreting germ of Döderlein. While it is incapable of infecting the in-

tact vulvar and vaginal surfaces, it thrives in the urethra, cervix and utricular glands. Its presence is usually due to ejaculated semen, but it may gain entrance to the vagina by innocent contamination. It has little tendency to permeate the lymphatics and seldom produces systemic poisoning. Whatever inflammatory reaction is present is due more to excreted toxins than to the presence of the germ itself.

Association with a mixed infection always adds to its virulence, but it has been found in pure culture in the tubes and in the peritoneal cavity. The gonococcus may invade the peritoneal cavity by continuity of structure, travelling along the mucous membranes, more rarely by the blood stream or lymph channels; though it has been demonstrated in the blood, endocardium, pleura, and peritoneum. The usual route of the gonococcus is by the mucosa. The gonococcus has the faculty of preparing the tissues for the invasion of other organisms, and by symbiosis exalts their virulence as well as its own, and thus becomes a potent factor in increasing the virulence of mixed infection. The gonococcus may remain latent for years in the crypts of the vulva, the ducts of Bartholin's glands, the urethra, Skene's tubules, and the cervical glands. The gonococcus produces no exotoxin, but contains an endotoxin which is leukotactic, yet the bacteria seem to do but little harm to the leukocytes.

The other important cocci infectors are the streptococcus pyogenes, and the staphylococcus aureus and albus, of which the staphylococcus aureus is the most common offender. These germs never originate "de novo" nor are they idiopathic, but their natural habitat is the cutaneous surfaces, mucous membranes, and dirt, such as may be found under the finger nails of the nurse or attendant. They enter the tissues through a wound or abrasion and usually invade by the lymphatics. They infect more quickly and with more virulence than the gonococcus.

The streptococcus was discovered by Mayerhofer in 1865, and isolated in pure culture by Ogsten in 1883. It may gain entrance at any point where solution of continuity has taken place in the genital tract, and passes at once into the lymph spaces and along the capillaries to the blood stream, without producing much local reaction (the virulence of the invader determines the amount of exudative resistance it excites). Consequently the serous or seropurulent exudate poured out may be much or little. The streptococcus, unless of minimal virulence, does not produce soluble toxins, but its body contains hemolysin and leukocidin, although the actual toxic agent is unknown. The various forms of streptococci are the longus, brevis, erysipelatis, capsulatus, and viridans. Hemolysis is not a constant characteristic and does not indicate excessive virulence, nor does the length of the chains, though it must be admitted

that long chains are usually the most dangerous. The streptococcus may be found on the vulva and in the vagina of many apparently healthy women. It exists in a state of very low virulence as a saprophyte, which, under proper environment and cultural influence, may become more dangerous.

The staphylococcus was separated from the streptococcus by Ogsten in 1883 in the pus from purulent infections; and Brieger in 1888 demonstrated the staphylococcus aureus in five fatal cases of puerperal infection. The staphylococcus occurs in various forms, e.g., the aureus, albus, flavus, and citreus. This entire group is frequently classified as pyococci, and they are less invasive than the streptococcus. They usually cause a greater leukocytic reaction, and by possessing leukocidin and proteolytic ferment, they rapidly kill the leukocytes, liquefy the tissues, and produce pus.

Local and general leukocytosis is characteristic in infection produced by this bacterium. The aureus and occasionally the albus, as demonstrated in four of the writer's cases, may be of a hemolytic strain. Severe and fatal bacteriemias may be caused by these organisms. These pyococci are found on every vulva and in most vaginas before labor and during the puerperium. In these locations they live as harmless parasites, unless their virulence is exalted by such accidental conditions as trauma, abrasions, and lacerations, which offer a port of entry for their latent energies. This is instanced in the suppurations found in perineorrhaphies and many of the parametric abscesses seen postpartum.

The bacillus coli communis is the chief organism inhabiting the large intestine. It closely resembles the typhoid bacillus and is associated with many pelvic inflammatory conditions, in which there are intestinal adhesions. It always migrates from the intestine through adhesion of the intestine to the visceral or parietal peritoneum. This germ has been isolated from pus in tubal and ovarian abscesses. It has the power to kill many of the cocci which infect the pelvic structures, and then, after rendering the contained pus free of streptococci, staphylococci, etc., it dies, leaving the contained pus sterile but impregnated with its characteristic odor.

In addition to the bacterial flora already described, mention may be made of the bacillus aërogenes capsulatus which was discovered by Welch in 1891. It is a non-motile, spore forming, encapsulated gram positive bacillus, which is an obligate anaërobe that will not grow in the presence of oxygen. It is commonly classed as a saprophytic organism, living only on dead tissue, but it produces a powerful exotoxin which is capable of producing further tissue necrosis and prepares the way for the

invasion of other organisms, especially the streptococcus. Its normal habitat is in the feces of humans and animals, and in the soil. It has occasionally been found in the dust of hospital wards.

Mode of Invasion.—Negelius has found the following types of bacteria more or less constant in the flora of the vulva, aërobic streptococci and staphylococci, the bacillus coli communis, anaërobic streptococci and staphylococci, and the bacillus capsulatus. If this last named organism is a constant inhabitant of the vulva and the feces are constantly pouring out its Welch bacilli, it is easy to explain how any woman may become infected through intravaginal manipulation by the gas bacillus. Fortunately, however, man is peculiarly resistant to the invasion of the Welch bacillus, for this organism does not easily develop and propagate in the body, unless it has some dead tissue to live upon. *All observers agree that the cavity of the uterus is free from bacteria during normal pregnancy,* but during the puerperium organisms undoubtedly ascend into the uterus from the vulva and the vagina, even in women who have never been examined. Hellendahl demonstrated that this may take place along the blood coagula which are usually present in the upper vagina and cervix.

Schottmüller claims to have isolated the Welch bacillus from the lochia of the normal puerperal woman. These facts show that the puerpera, with the bactericidal powers of her lochial blood during the first forty-eight hours, and her leukocytic zone after that, possesses one of the most wonderful protecting organisms against infection, of which we have any example in the body.

Krönig and Menge in 1907 showed that the liquor amnii may become infected with the gas bacillus prior to the rupture of the membranes. The writer has also seen and reported one case of gas bacillus infection at the eighth month, with infection of the fetus and physometra with membranes intact. *The vulva, vagina and cervix are the habitat of numberless non-pathogenic bacteria which are normal to these locations.* Döderlein isolated a vaginal bacillus with acid secreting properties, which he thought preserved natural vaginal acidity and inhibited the growth of pathogenic organisms. Krönig has described other non-pathogenic germs in the vagina, and Stroganoff noticed that during menstruation, vaginal micro-organisms increased in abundance. On the other hand, all observers admit that the uterine cavity and tubes are bacteria free in health. Recently Curtis, in a comprehensive bacteriological study of the endometrium in health and disease, concludes that endometritis *per se,* with bacteria present in smears or cultures, is practically unknown as a clinical entity, for, with the exception of pyometra and in instances

of recent exploration of the uterine cavity, the endometrium is almost invariably free from bacteria. It is conceded that, though these non-pathogenic bacteria in the vagina or around the vulva and vestibule are innocuous in these localities, *if they are introduced beyond the os externum and into a favorable culture medium by any agency, they become pathogenic.*

The vagina in the virgin is normally sterile; in the multipara however, with a relaxed or torn vaginal outlet, the lower part of the canal is covered on the surface with bacteria; but owing to the stratified pavement epithelium and the strong bactericidal power of the secretions present, pyogenic organisms which are introduced into the normally intact vagina are rapidly killed.

As has been stated, the uterus is normally sterile; but during labor, abortion, the puerperium and menstruation, the reaction of the vaginal secretion is changed by the addition of blood, mucus, etc., and becomes less resistant. The organisms which are ordinarily inactive multiply in this congenial soil, and when an avenue of entrance is made, as by trauma, infection follows, and of course this may extend to the uterus.

Septic infections which occur in connection with labor, abortion or the puerperium present features which are characteristic. The clinical course is determined, first, by the character, life history and habits of the infecting bacteria, and, second, by the anatomic conditions which exist during these periods. Hence, in considering pelvic infections in women, we necessarly have to study them in the following classes: (a) Puerperal infections, (b) non-puerperal infections, (c) gonorrheal infections.

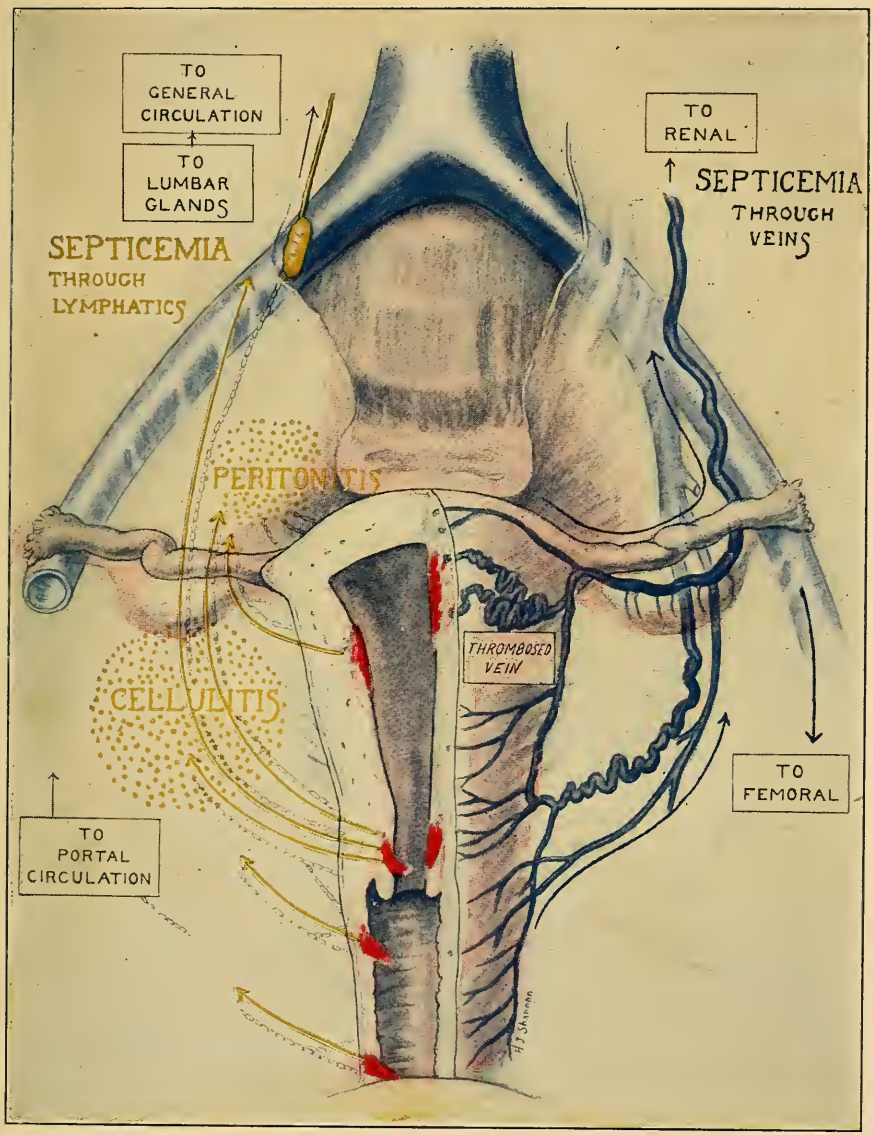

FIG. 21.—DIAGRAM ILLUSTRATING ROUTES OF PUERPERAL INFECTIONS FROM PUERPERAL WOUNDS THROUGH THE LYMPHATICS, PLACENTAL SITE, AND VENOUS RADICLES.

CHAPTER IV

PUERPERAL INFECTIONS

Puerperal infections—Avenues of entrance for bacteria—Birth traumatisms—Bacteria of the genital region—Primary focal infection—The puerperal endometrium—Puerperal endometritis—Frequency—Vulvar infection—Coccal endometritis—Cellulitis—Parametritis and perimetritis—Differential diagnosis of cellular inflammations.

Puerperal Infections.—Like any other infection depending on the inoculation of the puerperal wound by a bacterium, may be either local or general. Hence the infection may begin as (1) a wound inoculation, i.e., inoculation with the bacteria of any area where the normal reparative and healing process is taking place, with an associated toxinemia; this is similar in its pathology to the toxic absorption from the sapremia of the older writers; or to the toxic symptoms produced from lochiometra; (2) or, as a wound infection, which may be divided into (a) the local process illustrated in the infected perineum, cervix and endometrium, in which there is a tissue reaction which limits the extension of the infective processes; (b) the spreading of the infection beyond the wound area, which is due either to the increased virulence of the infecting bacterium, or the diminished resistance of the tissues. This spreading infection occurs through (a′) the blood vessels, in which case it may manifest itself clinically as a thrombophlebitis, pyemia, or bacteriemia, or (b′) through the lymphatics, producing parametritis, peritonitis, metritis desiccans, etc.

Avenues of Entrance for Bacteria—Birth Traumatisms.—Traumatisms of the birth canal which may occur during the course of labor include rupture of the uterus, lacerations of the cervix, vagina, vulva and perineum. *These are all contused and lacerated wounds, consequently the tissue resistance is lowered and bacterial inoculation and infective invasion are favored.*

Injuries to the cervix are generally recognized as among the most important factors in the production of puerperal morbidity, for these wounds act as the chief port of entry for the passage of bacteria into the parametrium. Traumatisms may therefore be considered as the principal avenues of entrance for the bacteria of the genital canal and other bacteria introduced from without.

Bacteria of the Genital Region.—In the great majority of cases the uterus and its contents are sterile before delivery, except in certain cases of acute endometritis and in putrefaction of the dead fetus and where the membranes have been long ruptured. A healthy vagina may harbor numberless pathogenic germs, which have migrated from the vulva, and yet these bacteria do not infect, unless the surface epithelium is broken by trauma. During labor and after delivery the bacteria which are harbored in the vagina may manage to reach the uterine cavity, where, under favorable conditions, they are destroyed by the bactericidal action of the uterine secretions. Ordinarily during the course of normal involution with proper uterine retraction and drainage, *the uterus is capable of sterilizing its cavity.* However, when the retraction and contraction are poor the contained bacteria not only multiply with amazing rapidity, owing to the retained blood clots which act as culture media, but many gain entrance to the uterine and para-uterine tissues, as has been shown by Sampson, through the lymph channels or venous radicles. Hence, the bacteriology of the vulva, vagina and uterine cavity in these cases is practically the same, for we simply have an upward migration of the vulva germs. Saprophytes and pathogenic cocci may occur side by side, and the saprophytes sometimes develop pathogenic qualities, while the pathogenic bacteria may play the rôle of saprophytes. Furthermore, it must not be forgotten that ordinary pyo-

Fig. 22.—A Relaxed Uterus After a Curetted Abortion. It Allows the Barium Solution to Be Forced into the Veins. (Sampson.)

genic cocci, acting temporarily as saprophytes or scavengers, may become virulent, and that ordinary putrefactive bacteria may in the presence of the proper culture media (as blood clots, pieces of placenta, membranes, etc.) multiply so rapidly on this dead material that they are capable of producing a severe toxinemia.

Pathology.—Infections first appear in the puerperium as (1) local infections, which may be primitive or consecutive, or as (2) blood states, including toxinemia, bacteriemia and pyemia. A local infection may or may not be associated with toxinemia or bacteriemia, while in the blood

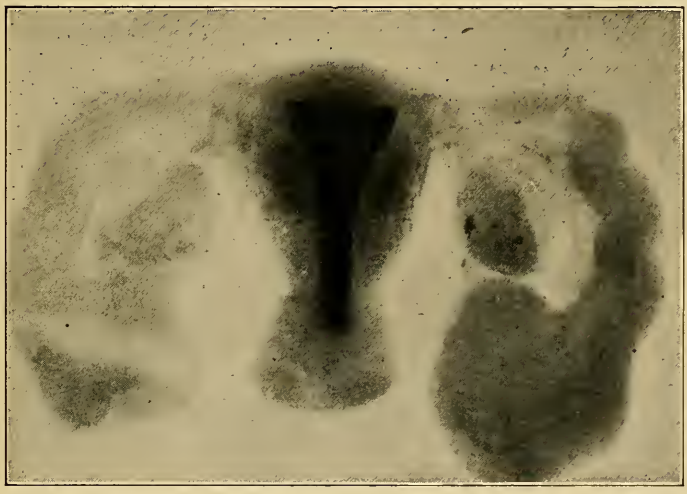

Fig. 23.—When the Endometrium Is Intact and the Uterus Contracted Barium Solutions Cannot Be Forced into the Veins. (Sampson.)

states a toxinemia may occur without bacteriemia. Local infections may be divided into those which are primary or primitive, and those which are consecutive.

Primary local infection is the direct inoculation of a wound by germs, while consecutive lesions result from the extension of the infection though the lymphatics or blood stream. As illustrative of primary local lesions, we have (a) puerperal ulcers, (b) endometritis. These lesions develop from the inoculation of germs into traumatized or wounded areas, as the inoculation of lacerations of the vulva, vagina, perineum, cervix, or the endometrium, which result in a puerperal ulcer, having the same pathology, whatever its location.

A puerperal ulcer is a simple wound infection and is usually met with in the form of infected lacerations of the vulva, vagina and cervix. These primary injuries are commonly lacerated wounds, which naturally tend

to heal by the normal process of wound repair; but if the vaginal secretion or lochia contains virulent pyogenic bacteria, wound inoculation takes place, and if the wounds have been closed with sutures, the sutures cut through, and suppuration takes place along the suture tract. *Infection of the wound excites in the immediate surrounding tissues a tissue reaction with the formation of a more or less complete defensive wall of leukocytes.* The ulcer is usually covered with a diphtheroid false membrane, due to the irritating properties of the bacterial toxins. If the leukocytic and tissue reaction is sufficient to prevent further bacterial extension, the *tissues immediately surrounding the ulcer become infiltrated with exudate* and the false membrane is exfoliated, leaving a granulating wound surface which is protective. During the tissue reaction there is always some toxin absorption, i.e., a mild toxinemia. It is also possible for a blood stream infection to have its source in the infection of a perineal or vaginal wound by hemolytic cocci, for cocci of this type excite little or no local protective tissue reaction. These ulcers are frequently associated with septic endometritis, in which case they represent only a minor complication. When, on inspection, the cervix is seen to be free from such ulcerative changes, it is usually conceded that there is no intra-uterine lesion.

THE PUERPERAL ENDOMETRIUM

The endometrium, after labor or abortion, should be considered as a traumatized wound undergoing the normal process of wound repair, and may be infected by pathogenic microörganisms, in which case it is virtually a large puerperal ulcer. It must not be supposed that the presence of necrotic decidua or even a piece of retained placenta within the cavity of the uterus will produce an endometritis. *In order to have an inflammatory reaction there must be infection.* Retained products of conception simply act as culture media for bacteria, and prevent proper retraction and contraction of the uterus, which in turn diminishes the normal protection of the individual against bacterial invasion.

The writer has repeatedly left pieces of membranes, and in many instances the entire placenta, within the uterus for days, without having an endometritis produced, at least there has been no constitutional reaction, and these ovular remnants have been cast off spontaneously and normal involution has then progressed naturally.

Unfortunately the practitioner is too often unfamiliar with many of the local changes which actually take place in the endometrium after the delivery of the fetus. During pregnancy the glandular layer of the decidua vera and serotina undergoes a fatty degeneration. The separation

of the placenta and the membranes takes place in this layer; hence, when the uterus is emptied at the termination of labor, its mucous surface is denuded. This leaves the connective tissue spaces in the basal membrane exposed, and at the placental site the surfaces are bared almost to the muscle. With this fact in mind, it is easy to understand *that at the close of labor the entire interior of the uterus is one large wound.* At the placental site, where the connective tissue spaces are even more deeply exposed, the sinuses contain superficial thrombi. These open into and protrude out into the uterine cavity and mark the only irregularity in the intra-uterine cavity. With the retraction of the uterus the decidua is invaded by white blood corpuscles; these form a protecting layer, similar to that which takes place in any other wound, separating the necrosing parts of the decidua from the healthy underlying granulation tissue.

If we were to inspect the interior of the uterus immediately after labor, we should find the endometrium thickened to from two to five millimeters. The surface is roughened with attached shreds of degenerating decidua, blood clots, and bits of fetal membrane. By the third day after delivery, as the result of fatty degeneration, these surface structures have become so softened that they can be wiped off with the finger. *Nature, however, accomplishes this exfoliation by the development of the granulation wall which separates the dead from the living structures.* The remaining decidual cells return to their original condition. The remnants of the utricular glands covered with epithelium, which lie undisturbed among the muscle fibers, are brought closer together as the uterus retracts and diminishes its bulk, and the gland epithelium, under the stimulus of the circulatory stasis thus produced, proliferates rapidly and grows out on the surface of the endometrium, thus forming the epithelial covering of the new mucosa. Hence it will be seen that the greater part of the endometrium is cast off, and that its regeneration takes place from the connective tissue of the mucous membrane and from the gland epithelium, which proliferates from the deepest portions of the utricular glands. The changes therefore, which actually take place, differ in no way from the changes which occur in the healing of a granulating surface on any mucous membrane in any part of the body.

Puerperal Endometritis.—Pathology.—If infection of the uterine wound takes place by migration of infecting cocci from below or by their introduction from without, this normal process of wound healing is disturbed and the mucous membrane becomes swollen, rough and covered with the thickened decidua in a state of necrosis. The surface grows moist and is covered with a mucopurulent or mucosanguineous smeary material. The cervix is almost always coincidently enlarged, is swollen

and eroded, and if lacerated, the wounds are covered with a diphtheroid membrane. Uterine involution is arrested because of the reaction which is excited in the subjacent tissues and the consequent circulatory stasis

FIG. 24.—WHEN THE ENDOMETRIUM IS INJURED THE BARIUM SOLUTION MAY BE FORCED INTO THE VEINS. (Sampson.)

induced. The uterus becomes edematous and relaxed, and this gives the bacteria ready access to the blood stream through the venous radicles, for the terminal sinuses empty into the arcuate veins and they in turn into the uterine veins. Retraction of the uterus presents a barrier to bac-

FIG. 25.—VEINS INJECTED WITH BARIUM SOLUTION. (Sampson.)

terial invasion, conversely relaxation allows free access to the blood stream.

Bumm describes two distinct forms of endometritis, first, that which is due to putrid or bacillary infection, in which the bacteria are limited to the surface and to the necrotic material lying thereon *by the pro-*

tecting bank of leukocytes and granulation tissue; and second, a coccal or septic form, where the streptococci or pyococci invade the lymphatic blood vessels *by passing through the protective wall.*

Unfortunately this clean cut pathologic description is not borne out clinically, for it is seldom that the bacteria are wholly confined to the decidual surface, as there are many areas in which the protective zone is not so well defined as to prevent bacterial invasion into the underlying tissues.

In the putrid form the surface endometrium is covered with thick fetid material, often containing gas bubbles. The surface is of a greyish or yellowish or greenish color, or may even be black, because of the decomposed blood. It is impossible to wipe this surface smooth, as in the normal uterus, owing to the varying depth of the superficial necrosis. If the bacteria are virulent, there may be actual sloughing of the endometrium; so that, instead of having a smooth, granulating surface, we find patches of localized ulceration and the whole interior of the uterus may become putrescent. This is the case when the putrefactive bacteria are acting jointly with pyococcic germs. If this superficial gangrene goes still deeper into the uterus, owing to the greater virulence of the bacteria, portions of the lining of the uterus, and even some of the underlying muscle itself, may slough off, producing the metritis desiccans described by Garrigues. *If the infection is due to the streptococcus pyogenes and pyococci alone, unmixed with saprophytes, there is no fetor, and the surface of the interior of the uterus is usually smooth and not deeply necrotic.*

While it is usual for the placental site to participate as a part of this whole infective process, *occasionally the infection is limited to the placental site,* and there is little local reaction except at this point. From this origin it is possible to have severe thrombophlebitic extension.

From what has been said it will be seen that, in a large majority of cases, the bank of granulation tissue and leukocytes is sufficient to limit the infection to the interior of the uterus, *unless nature's efforts are interfered with by the meddlesome obstetrician, who insists that, because there is necrotic material within the uterus, even though it be the result of nature's conservative starvation process, he must remove it and by so doing break through the barrier which nature has placed there to protect the organ against the infecting organism.*

The infecting organism may be a saprophyte or pyogenic coccus, or both may be present and here again, dependent upon the tissue resistance and the virulence of the infecting cocci, we may have a simple localized infection, in which the formation of a phagocytic barrier and the occlu-

sion of the placental sinuses protect the contiguous structures and the general organism from the extension of the disease. When there is a mild constitutional reaction, like the simple surgical fever following operation, i.e., toxinemia, or, on the other hand, when the microörganisms break through the protective zone and invade the peri-uterine tissues by the lymphatic route, or penetrate into the venous radicles and uterine veins, the constitutional intoxication becomes severe, at times violent.

Frequency.—Even before Lister's great discovery, obstetricians recognized that puerperal fever was a wound infection, almost always conveyed from without by hands or instruments. Kirkland in 1774 wrote describing its contagiousness, while Gordon of Aberdeen in 1795 demonstrated that the disease was always conveyed from one patient to another by physicians or nurses. Semmelweis in Vienna in 1846 found that the mortality of labor cases attended by students engaged in post-mortem work and dissecting was four times greater than that of those cases attended by the midwives in the clinic. By simply having the students thoroughly wash their hands he reduced the mortality of the clinic from eleven per cent to one per cent.

In hospital practice, where every detail in aseptic care is given the pregnant and parturient woman, the mortality and morbidity from puerperal septic infection has been brought down to an almost irreducible minimum. In 5000 consecutive confinements attended by the senior students of the Long Island College Hospital in the outpatient service, there have been no deaths from infection. These women have all had prenatal attention in our clinic, and have been delivered in their homes by employment of a simple aseptic technic. No vaginal examinations were made, but the progress of labor was watched throughout by abdominal and rectal examination.

The total morbidity in this series was less than three per cent. In our morbidity records we include all cases where a temperature of 100.8° occurs after the first forty-eight hours and such elevation persists for twenty-four hours. On the inside maternity service the morbidity has been 9.3 per cent. This includes naturally many emergency and operative cases, which have had vaginal examinations through an unclipped vulva before admission to the hospital; yet our mortality in the past five years has been less than one half of one per cent. Contrast this with the Health Department records of the mortality in private practice.

Dublin, in a statistical review of 14,694,260 women of child bearing age between fifteen and forty-four, found the total death rate from diseases incidental to pregnancy, parturition, and the puerperium, to be 68.4 per 100,000, and 43 per cent of this total was from puerperal infection.

Such statistics show how little all of our antiseptic and aseptic propaganda has done to improve the technic of the general practitioner.

The midwife is often blamed for infection in private practice, but an extended experience in consultation work has convinced us that the ordinary foreign trained midwife is not a menace, but rather a boon to the parturient woman. Would that we could, as Williams suggests, train our practitioners to be contented to be clean male midwives, and not pose as operative obstetric surgeons, without having had any special training.

Puerperal fever is primarily due to infection of the obstetric wound by microörganisms. The entire parturient canal following delivery may be regarded as a wounded surface, and is therefore a suitable gateway for the entrance of infective bacteria. The uterus, however, with its bruised cervix and placental site, must be considered the chief port of entry. *Infection may start in wounds of the vulva, vagina, cervix, or in the uterus itself.*

Vulvar Infection.—Infected wounds in this location present the same general appearance as septic wounds elsewhere. In severe cases they are of a dirty greenish yellow color, bathed in a foul purulent discharge, and called puerperal ulcers.

Septic wounds of the vagina after instrumental delivery are relatively common. It matters not whether the injury results from natural or instrumental delivery, *the wound is a lacerated one in bruised tissues,* which has diminished resistance; hence, it is easy for infection to extend through these wounds, beginning in the pelvic floor and vagina, and producing in the surrounding cellular tissues paravaginal inflammation.

Rupture of the vagina, with extension of the rent into the cellular tissue, peritoneum or bladder, is becoming more and more rare as the forceps is less frequently used; yet, a superficial vaginitis with purulent discharge is a common postpartum sequela.

The cervix in every labor is subjected to severe trauma and some degree of laceration, and these wounds may become infected and the infection may extend into the cellular tissues surrounding the uterus, or may reach the peritoneum or the blood stream by way of the lymphatics and blood vessels. A posterior parametritis which is very obstinate is not an uncommon sequel to cervix infections, and explains many of the postpartum backaches. The uterus however must of necessity, either through lacerations, or through its endometrium or the placental site, be the chief port of entry.

To properly understand puerperal infection of uterine origin, one must consider, for a moment, the physiological process which takes place

within the uterus after the evacuation of its contents as a consequence of the rapid retraction and resulting hemostasis. This sudden shutting off of the blood supply leaves the endometrium more or less covered by a layer of necrosing decidua, which is cast off piecemeal by a process of surface starvation, due to the active leukocytosis and the increase of the connective tissue cells which take place immediately beneath the decidua. This forms a granulating zone which prevents the invasion of all but the more virulent bacteria. The entire cavity of the uterus, except the placental site, becomes a granulating wound by about the end of the first week, protected, as is the case in the ordinary surgical wound, by its granulating surface. In the placental site the thrombi in the open vessels require a longer time for this organization. This site is rough and raised, and is covered with irregular masses of decidua, shreds of placenta, fragments of villi, etc. Organization occurs in the thrombotic sinuses, while the bank of granulation thickens and causes the exfoliation of the superimposed masses. This occurs from the tenth to the fifteenth day. The effect of infective bacteria on these normal healing processes varies with the nature and virulence of the microörganisms and the manner by and the soil into which they are introduced.

Any of the infecting organisms, singly or in combination, such as the various strains of streptococci, the staphylococcus, pneumococcus and gonococcus, as well as numberless non-pathogenic bacteria, may be found in the postabortal or postpartum uterus. Repeated culture of the interior of the uterus at various periods of the puerperium shows that its contents after the first twenty-four hours postpartum are seldom sterile; *in fact over 50 per cent of the cases which were cultured in our clinic on the fifth and sixth day postpartum showed the presence of infective bacteria within the uterus, yet these patients did not have sepsis, for the granulation zone was intact.* Puerperal and postabortal infection of the uterus must, therefore, be regarded as a wound infection with an absorption of toxins. Infection of the puerperal wound is no different in its pathology from an infection of any wound on the surface of the body, except that the area of lymphatic drainage is greater.

Notwithstanding the presence of infective bacteria in the uterine cavity, the uterus is normally protected from invasion from the interior both during and after labor. During the labor it is protected by (1) the membranes, though in recent years Selmons and the writer have shown that *bacteria can penetrate the amnion, if the labor is prolonged and the membranes have ruptured early,* owing to the changes in the amniotic endothelium, which diminish its resistance and allow bacterial invasion of the placental site; also by (2) the autotoxic properties of newly let

blood. After labor the uterus is protected by the establishment of the lochial discharges and the normal reaction in the uterine tissues. This is shown by its protective granulation zone, composed of polynuclear leukocytes, eosinophiles, fibroblasts and polyblasts. *Infective organisms must penetrate this zone or pass through abrasions or other injuries in the uterine tissues to get outside of the uterus.* This the streptococcus and some strains of the staphylococcus have the power of doing. The gonococcus, on the other hand, lacks this power of penetrability, and advances along the mucous membrane and submucous lymphatics and reaches the tubes and peritoneum by continuity.

The primary inflammatory lesion in puerperal and postabortal infection, if the infecting cocci are not of the hemolytic type, is found within the uterus. *Endometritis is the primary lesion* in most cases, as seen by the following enumeration of factors which favor uterine infection and the development or spread of infective organisms.

1. The cervix and lower uterine segment are particularly exposed to traumatisms of labor and to injury in the course of operative delivery.

2. The cervical and uterine mucous membrane is a very delicate structure as compared with that of the vagina, and is covered with a layer of dying decidua, which acts as a favorable culture medium to the growth of bacteria.

3. Blood clots, membrane, and even placental fragments may be retained within a relaxed uterus, and these also may act as culture media for pathogenic bacteria.

4. Infection may have been present in the endometrium during pregnancy or have been brought there directly by the introduction of a colpeurynter or bougie or by decomposed liquor amnii.

5. Shreds of membrane from the uterus, hanging down into the vagina, may act as a bridge on which bacteria from the unsterile vagina may mount into the uterus.

6. The open vessels of the placental site are particularly favorable to the migration of germs.

7. If the uterus is flabby and relaxed, drainage is impaired and stasis of the lochia may occur, and this produces a condition of lochiometra which is particularly favorable to bacterial growth. While all of the above mentioned conditions predispose to intra-uterine infection, *we know from clinical experience that the presence of decidua or a piece of placenta retained within the uterus will not cause endometritis unless infection is also present.*

While making our clinical investigations on this subject, we have left pieces of placenta in the uterus for weeks without harm or decomposition,

so long as infection was not introduced by careless examination or intra-uterine instrumentation.

A well contracted uterus and the Fowler position, the elevated trunk posture, alternating this posture with the patient lying on her abdomen to drain the vagina, will expel decomposing clots, placental fragments, and retained secretions. Firm contraction and retraction also offer a barrier to bacterial invasion from the uterine cavity, by closing the lymphatics and blood vessels. Sampson has shown that in abortions an intact endometrium will protect the venous radicles in the uterine wall from the invasion of intra-uterine bacteria. On the other hand, when a relaxed uterus becomes bent upon itself, it may be caught behind the pubis and become extremely anteflexed, or may be caught below the promontory and become retroflexed, and so prevent the free outflow of the lochia, thus producing lochiometra and favoring the absorption of toxins. *From the clinical facts we must conclude that a well contracted uterus, in normal anteversion, is capable of emptying itself of its content, if infection is not introduced from the outside.*

Fig. 26.—Barium Injected into the Contracted Uterus Can Be Forced into the Tubes. (Sampson.)

Fig. 27.—Relaxed Uterus with Tube Injected with Barium Solution. (Sampson.)

Putrid or saprophytic endometritis is an infection of the dead and necrotic superficial structures retained within the uterus, which produces

irritant material composed of bacterial toxalbumoses and ptomains, which in turn irritate the endometrium and excite a tissue reaction.

These necrotic structures have bacteria *in them* or *on them*. Schottmüller showed that the majority of cases of endometritis putrida were due to an obligate anaërobic streptococcus. In this form of infection the uterine tissues are protected from further invasion by the presence of the granulation zone.

The ptomains and bacterial toxalbumoses set up an endometritis by exciting round cell proliferation in the deeper layers of the endometrium, which ends in a superficial necrosis of the overlying tissues. The degree of this necrosis depends in part upon the contraction and retraction of the uterus. *If the uterine content is evacuated within a reasonable time,* extensive necrosis of the endometrium does not result; but if the tissue reaction continues and the round tissue cells become banked up beneath the endometrium, the necrosis is extensive. If the necrosis is slight, we have only an intensification of the normal exfoliation of the mucosa. Thus, with the cause removed, the emigration of phagocytic leukocytes and the formation of bactericidal lochia tend to cleanse the uterine cavity. If, on the other hand, the necrosis is considerable, it may interfere with the normal regeneration of the endometrium. Hence, with the uterine cavity open to the migration of pathogenic cocci from below, a mixed infection develops and the patient may succumb from the severity of the absorption; for saprophytes have been found in the blood during life, while pyogenic cocci are known to have a definite penetrability.

Symptoms.—When this condition exists within the uterus, there is found a definite train of characteristic symptoms and signs. *The lochia remains bloody, excessive, and fetid, and is frothy* from an admixture of gas bubbles. An examination of the secretions shows the presence of saprophytes and cocci of low virulence. *The after pains continue* and from time to time a clot is expelled by painful uterine contraction. *There is a toxinemia* from the absorption of the toxins produced by the superficial necrosis; *this absorption from the uterine cavity causes an elevation of temperature and a slight acceleration in the pulse rate.* Abdominal examination will show that the *involution of the uterus is retarded, the uterus being large, tender and more or less relaxed.* Should the pelvis be digitally explored, the cervix will be found open, swollen and eroded, and if the gloved finger is passed into the uterine cavity, clots and necrotic débris are encountered and the interior surface of the uterine cavity is rough and shaggy.

Prognosis.—The establishment of proper uterine drainage, inducing retraction and contraction of the uterus, promptly controls this type of

lesion and the fever subsides. Hence it will be argued, if expulsion of the contents is followed by a prompt subsidence of the symptoms, why not empty the uterus of this necrotic débris by surgical methods?

Experience has taught us that any sort of trauma to the delicate granulation wall, which is confining the infection within the uterus, opens avenues of extension, and that lateral prametritis is a constant sequel of attempts at digital or instrumental evacuation. It does no harm to remove sterile contents, but manipulation always spreads infection when the content is already infected.

Coccal Endometritis.—The second type of infection met with within the uterus may properly be called a coccal or pyogenic endometritis; in this form the infective bacteria, the streptococcus or other pyococci have more marked invasive qualities and attack the living tissues, and are introduced or penetrate into the lymphoid lining, or the myometrium, causing a prompt tissue reaction and producing a necrotic layer of endometrium, which resembles the false membrane of diphtheria.

Whether these cocci advance further than the interior of the uterus and invade the lymphatics and blood vessels, or remain confined within the uterine cavity, depends on the completeness of development of the granulation zone and the virulence and the penetrability of the invading bacteria. If the reaction is sufficient to excite prompt resistance and the leukocytic barrier increases in thickness, the lochia acquires bactericidal properties, which tend to sterilize the interior of the uterus. This observation has been checked up numberless times in our clinic. With *every puerperal endometritis there is always an associated metritis;* this is a defensive reaction on the part of the myometrium against the invading cocci. In this reaction small round tissue cells, leukocytes, fibroblasts, and polyblasts are thrown out and deposited between the muscle fibers and around the gland tubules, and the further extension of the cocci is halted. From this primary endometritis and metritis the bacterial invasion may extend through the lymphatics in the uterus into the surrounding connective tissue, or to the peritoneum or through the veins in the placental site to the blood stream.

Since the infection often begins at the placental site, much depends on its condition at the time of exposure. If the uterus is well retracted and the sinuses are closed, the defense at this point is effective; on the other hand, if the sinuses are simply plugged with aseptic thrombi, virulent cocci like the streptococcus may infect these thrombi directly or penetrate between the sinuses and enter the vessels from the outside and thus gain entrance to the circulation.

Infections due to the streptococcus pyogenes and the pyococci alone

do not give rise to fetor, and the interior surface of the uterus is usually smooth and not deeply necrotic. As a rule the bank of granulation tissue suffices to limit the infection to the uterus, unless *nature's beneficent processes are disturbed by the meddling of the accoucheur.*

Symptoms of Coccal Endometritis.—For the first two or three days the puerpera is fairly comfortable, but there is usually some indication of brewing trouble, such as malaise, a higher pulse rate than is normal, restlessness, or pain in the uterus, or *prolonged after pains.* On the third, fourth, or fifth day there is a slight chill or chilly sensation with a rise of temperature, headache, anorexia, and the patient is conscious of a feeling of heat over the body. The pulse may range from 100-140 and the temperature from 101-104° F, depending on the severity of the infection. The abdomen may become slightly distended, but there is little or no tenderness except over the uterus and the involution of the uterus is always retarded. If, however, the infection extends through the myometrium to the perimetrium, there is tenderness over the uterus and in both inguinal regions. The lochia are at first unaltered, but within forty-eight hours they have lost their characteristic qualities and become serous, flesh colored, or seropurulent. *The lochia are not foul, unless large numbers of saprophytes are developed.* The lochia, however, have caustic infective qualities and the wounds in the vagina and about the vulva, which are bathed in them, are covered with a pseudodiphtheritic membrane. On physical examination, the cervix is closed and the uterus fairly well retracted, and unless there has been parametrial extension its mobility is not interfered with. Were it possible to make a digital exploration of the interior of the uterus, it would have been found smooth, and the endometrium bathed in purulent or sanguinopurulent discharge, free from odor. Lymphatic invasion from the cervix is shown in the parametritis postica so commonly found postpartum, which is the chief cause of the backache experienced in the puerperium. Extension of infection from the cervix and higher up in the uterus is generally through the lymphatics in the broad ligaments. This extension produces a lateral parametritis or cellulitis.

Cellulitis.—Pelvic cellulitis or *parametritis* is an inflammatory reaction of the pelvic cellular tissue to a bacterial invasion. The bacteria reach the parametrium through the lymph stream, and excite a tissue reaction in which serum, leukocytes, and round tissue cells are poured out, producing a local inflammatory swelling.

Location of the Cellular Tissue in the Pelvis.—In order that we may better appreciate where to look for these inflammatory swellings, it may be well to briefly review the anatomic arrangement of the pelvic

connective tissue. The pelvic connective tissue lies under the peritoneum and between the pelvic peritoneum and the pelvic diaphragm. It forms the loose connecting and supporting areolar structure between the organs and the pelvic wall, and between contiguous viscera and soft structures. It spreads from the uterus as a center, and radiates outward in all directions, each part reaching the pelvic wall. It surrounds and supports the blood vessels, nerves, and lymphatics, and forms thin sheaths. It is con-

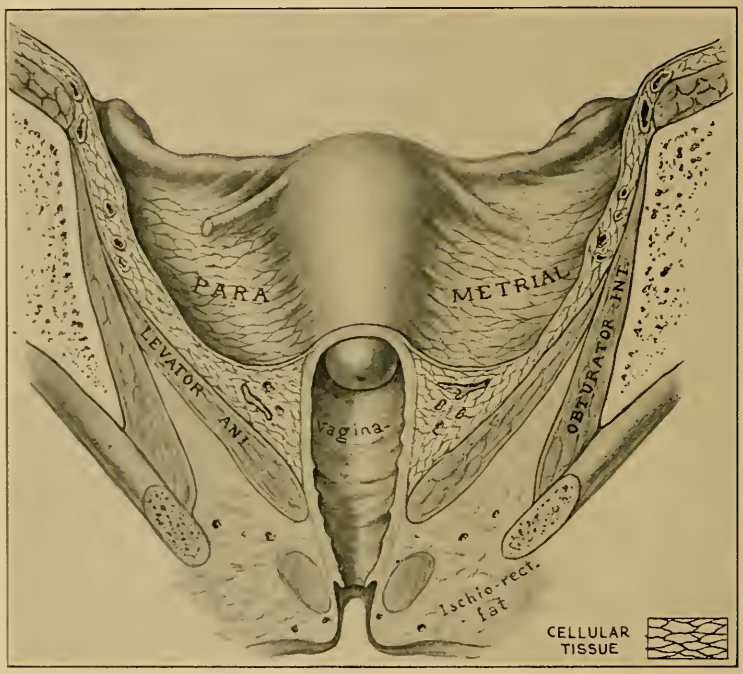

FIG. 28.—SHOWING THE CELLULAR TISSUE LYING UNDER THE PELVIC PERITONEUM AND ABOVE THE LEVATORES ANI MUSCLES.

densed into strong bands and ligaments, forming the aponeurosis of muscles and the ligamentary attachments of the pelvic viscera.

Infections from traumatisms of the vagina and cervix chiefly involve this loose areolar and fatty tissue, and the infection is directed by and confined between the fascial sheets and ligamentary planes. The subserous connective tissue may be traced across the pelvis from front to back, as this tissue is but a continuation of the subperitoneal layer of the abdominal wall. As the peritoneum passes off from the abdominal wall, it is closely applied to the fundus and posterior wall of the bladder; but anteriorly, between the bladder and the symphysis, there is a larger areolar tissue space (the space of Retzii). This loose tissue extends in a

thin layer along the anterior pelvic wall and over the fascia, covering the obturator internus muscle.

Between the posterior wall and the base of the bladder and the cervix uteri and vagina, is a loose connective tissue layer which allows the bladder to be easily stripped off from the front of the uterus and vagina. There is little loose cellular tissue at the fundus and down the back of the uterus, where the peritoneal attachment is firm.

Under the peritoneal covering of the cervix and posterior vaginal

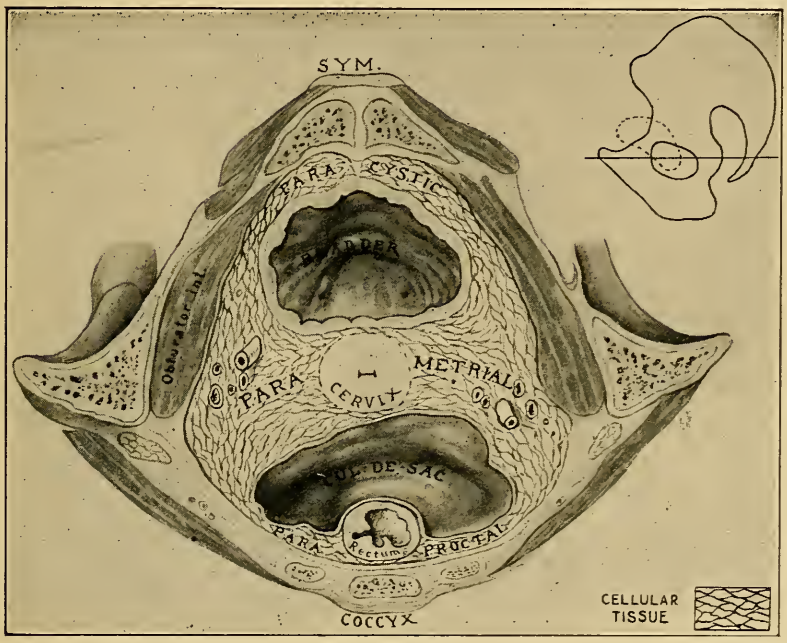

FIG. 29.—TRANSVERSE SECTION THROUGH PELVIS AT THE LEVEL OF THE INTERNAL OS, SHOWING HOW THE CELLULAR TISSUE SPREADS FROM THE UTERUS AS A CENTER, EACH PART REACHING THE PELVIC WALL.

wall a thin layer of connective tissue may be traced, having extensive connections with the retroperitoneal tissue at the back of the pelvis; this continues backward to the sacrum in the two uterosacral folds. There is also connective tissue at the sides of the rectum and in the mesentery of the pelvic colon. In tracing the cellular tissue across the pelvis from side to side, we find it extending from the pelvic wall to the uterus within the folds of the broad ligament, and from the uterus to the opposite pelvic wall. At the base of the broad ligament it is condensed into the utero-pelvic ligaments, which make one of the main supports of the uterus.

A great increase in the amount of pelvic cellular tissue takes place during pregnancy; this explains not only the frequency of parametritic

or cellulitic inflammations after labor, but the ease with which the pelvic organs are separated in operative procedures, i.e., the evidence of lines of cleavage.

The lymphatic channels, which drain the greater part of the vagina, the cervix and lower uterine segment, pass out along the base of the broad ligament, and are supported by this arbor of cellular tissue. These follow the course of the uterine vessels to the hypogastric and iliac glands.

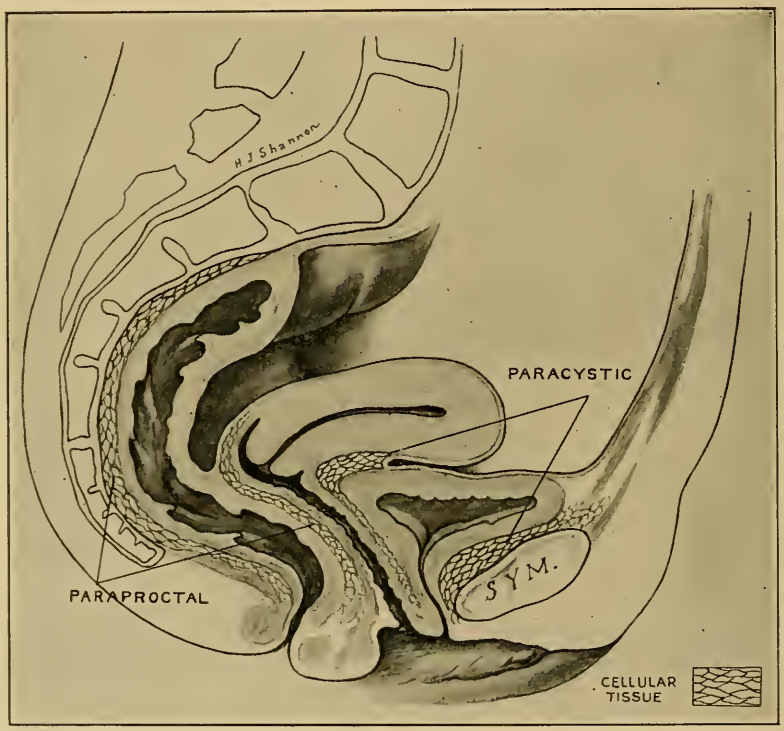

FIG. 30.—SAGITTAL SECTION SHOWING THE RELATION OF THE PELVIC CELLULAR TISSUE TO THE BLADDER AND THE RECTUM.

The lymphatics of the fundus and upper part of the body of the uterus follow the ovarian vessels in the infundibulopelvic ligament to the glands at the bifurcation of the aorta and the lumbar group. Lymph channels also run in the uterosacral ligaments to the sacral glands, and through the round ligaments to the inguinal glands.

ETIOLOGY.—The majority of cases of cellulitis are due to infection by the streptococcus pyogenes. The staphylococcus and bacillus coli, and occasionally the gonococcus, are found in combination, but the streptococcus is the chief infecting agent of cellular tissue.

The severity of the infection depends on the virulence of the infect-

ing organism, and the individual resistance of the patient, rather than the variety of the organisms. It has not been proven that the gonococcus can by itself produce primary pelvic cellulitis, neither does an uncomplicated gonorrhea give rise to the same inflammation and abscess formation seen in a streptococcus infection.

The most common avenue of entrance is through injuries to the cervix and vaginal vault during labor; for, besides the general softening of the tissues and enlargement of the connective tissue spaces, and the increased vascularity due to pregnancy, *there is a direct bruising of the parts by labor, all of which favor infection.* The amount of cellular tissue in this particular region is considerable, and the chief lymphatic channels draining it are found at the base of the broad ligament.

The cervix and surrounding tissues are subject to the greatest trauma, consequently the tissue resistance here is lowest; *furthermore, lacerations at these points open into extensive cellular spaces.* Even trivial injuries may act as the points of ingress, but as a rule there is the history of an instrumental delivery, manual or bag dilatation, or a dry labor with frequent vaginal examinations. The chances of infection are greater under these circumstances. On the other hand, *this form of inflammation is comparatively rare after abortion,* as the cervical tissues are not subjected to such a degree of trauma; hence tubal, rather than parametrial complications with peritoneal extension are the usual course. This is accounted for by the fact that the chance of injury during the passage of a small ovum is less, and the amount of connective tissue is also less than at full term.

Operations on both the pregnant and non-pregnant uterus, which involve opening up of the connective tissue planes, are frequently the cause of postoperative cellulitis. As illustrative of this, amputation of the cervix and hysterectomy by the Wertheimer method are frequently followed by large parametrial exudates.

Pathology.—The organisms invade the lymphatic channels and by their presence and the toxins they produce excite a hyperemia, which is followed by an effusion of protective serum, and a hurried migration of leukocytes into the soft areolar tissue, which, with the deposition of small round cells, make up the exudate. This increases the tissue bulk and gives rise to a soft swelling, which later becomes hard from the formation of a more fibrinous exudate. This exudate is generally limited at first to the base of the broad ligament on the involved side. As the exudate is poured out, it usually follows the line of least resistance in the cellular tissue between the fascial sheets forward and outward to the anterolateral pelvic wall and iliac fossa, or backward along the uterosacral

folds, lifting the posterior layer of the peritoneum. The fibrinous deposit which is thrown into the pararectal and prevertebral connective tissues, fixes and displaces the uterus and rectum, and more or less obliterates the portio, holding the pelvic organs in a hard, sensitive mass; or, the exudate may spread forward at the base of the bladder, and so reach the anterior pelvic and abdominal walls.

Clinically, we have found that the exudate may spread in almost any direction along the cellular tissue planes. It may be unilateral or bilateral, most frequently the former, or it may spread around the cervix

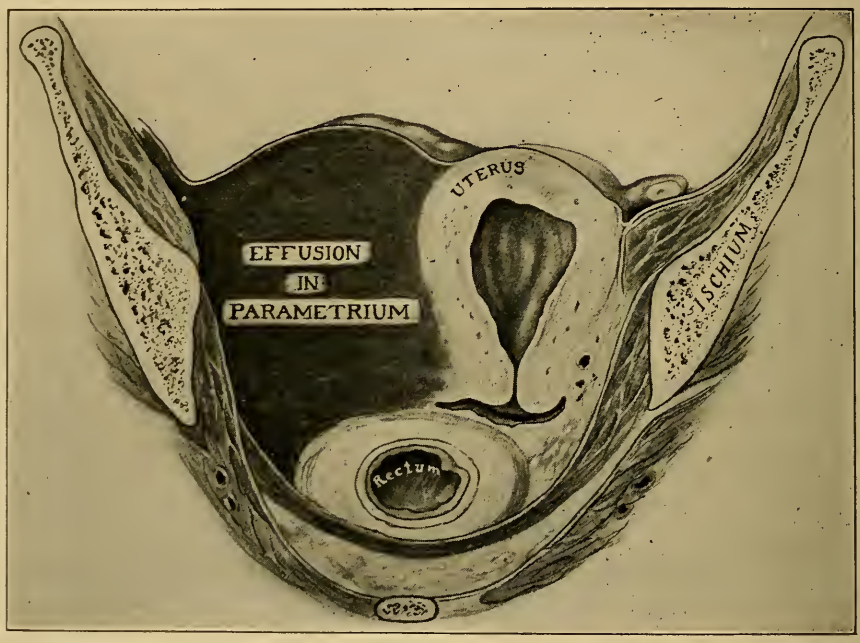

FIG. 31.—EXUDATE INTO THE LATERAL PARAMETRIAL TISSUES, DISPLACING THE UTERUS AND OBLITERATING THE FORNICES AND VAGINAL PORTIO.

from side to side obliterating the vaginal portio, leaving the os as a mere dimple in the vaginal vault; or the bacteria may follow an unanatomic course, even passing through muscle or fascia, in which case the exudate may be found in locations where it is least expected.

Von Rosthorn has classified pelvic exudates in the cellular tissues in a most practical way, showing their origin and their usual course of extension; his classification is as follows:

1. Those which come from bacterial invasion of cervical tears are usually lateral, horizontal exudates located in the base of the broad ligaments, and have a tendency to spread to the side walls of the pelvis and around the cervix.

2. Where the infective extension comes through the lymphatics of the uterus, usually from an endometritis. The exudates occur as high intraligamentous infiltrations, beginning near the uterine cornua, forming exudative tumors, rounded above with a tendency to unfold the broad ligaments, and extend into the iliac fossa.

3. Exudates into the retrocervical connective tissues from infective cervicitis. These spread posteriorly along the uterosacral ligaments, occasionally involving the sacro-iliac joints, or sink down into the connective tissue of the rectovaginal septum.

4. Exudates in the pericervical tissues spreading toward the sides of the pelvis, along and about the ureters.

5. Exudates anterior to the bladder which tend to spread up the abdominal wall behind the recti, forming abscesses in the abdominal wall, frequently due to the migration of the colon bacillus from the adherent intestinal masses.

The most common is the lateral exudate extending from the side of the uterus to the bony pelvic wall and then anteriorly, raising the peritoneum upward and appearing as a hard tender mass above Poupart's ligament. Rarer extensions are those travelling posteriorly to the sacro-iliac joints, and retroperitoneal extension upward toward the region of the kidney.

The writer has also seen extensions through the sacrosciatic foramen into the thigh, with abscesses posterior to the trochanter, and along the infundibulopelvic ligament to the psoas muscle.

In a recent case the bacterial invasion extended through the broad ligament, through the cellular tissue in the hilum of the ovary, and infected its stroma with a resulting abscess. Coincidentally, the infection passed through the sacrosciatic foramen and formed an abscess just posterior to the head of the femur. *Cul de sac* drainage and incision posterior to the trochanter temporarily controlled her sepsis, but not until the ovary with the contained abscess was removed did the patient recover her health.

The exudate varies in its extent and consistence, depending on the virulence of the germ and the resistance of the patient. In mild cases there may be nothing but a simple inflammatory edema, and again in the more virulent types of cellular infection the exudative process is limited to a serous and poorly defined cellular infiltration, for the bacteria quickly pass through the lymphatics to the peritoneum or into the blood stream. *Fortunately for the protection of the individual, in most cases there is an adequate protective tissue reaction with the formation of large exudates.* Section through these masses shows the lymph vessels

thickened, tortuous and beaded, and a yellowish or whitish pus exudes from numberless minute openings. The lymphatic chains are surrounded with exudate, giving them a glistening, glassy, moist appearance. The veins are often thrombotic, either from primary infection, or they may become secondarily infected and the thrombi may undergo puriform degeneration, the débris breaking up and getting into the circulation, forming infected emboli.

As the exudate increases in amount the blood supply is increased. This is especially apparent on the venous side, and later as cicatricial tissue

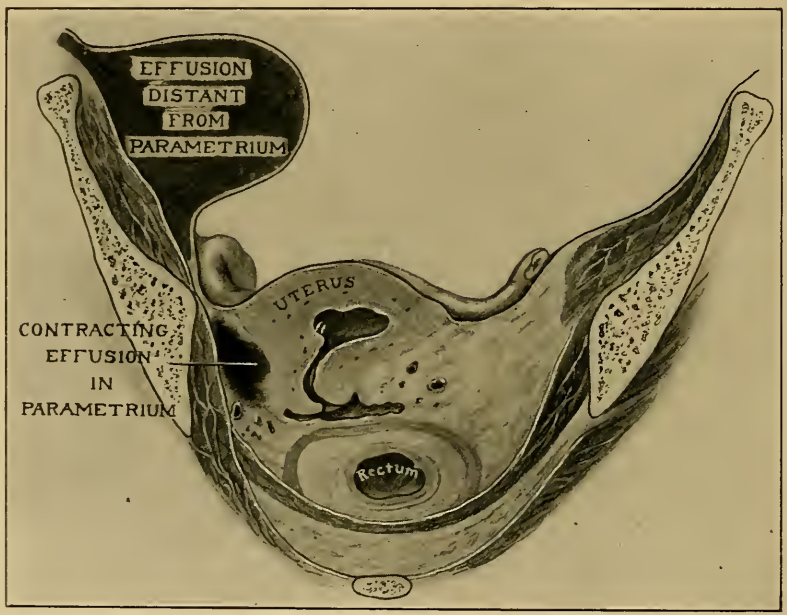

Fig. 32.—As the Exudate Organizes and the Scar Tissue Contracts, the Uterus Is Drawn Toward the Side of the Exudate.

forms and the scars shrink; the arteries are kinked and varicosities occur in the veins, while the ganglia and nerves may become pinched in the contracting cicatrices. This explains the pain and the frequency of pelvic varicosities in patients who give a history of an infected puerperium. Coincident with the pouring out of exudate into the cellular tissues in the broad ligaments subperitoneally, there is an edema, and necessarily the pelvic peritoneum takes part in the inflammation and throws out an exudate upon its surface, which causes the tubes and ovaries to become matted together and adherent to the broad ligaments, uterus, or the intestines, clinically giving the impression of large exudate masses.

Consequently it may be stated that parametritis always excites some

degree of perimetritis. This inflammatory exudate may undergo complete absorption, or may go on to suppuration. If absorption occurs, there is always some pathology which permanently remains. *When an exudate suppurates, the pus is discharged externally* or becomes encapsulated, limiting the mobility of the pelvic viscera and occasioning premenstrual pain.

In the milder infections with a serofibrinous exudation complete resolution usually takes place. Large masses of fibrinous exudate may completely disappear without leaving much edema or tissue damage. There are, however, always varicosities of the pelvic veins to tell the story of the intense venous engorgement, which was necessary to supply the protective exudate needed in nature's attempt to bury the infecting invaders (Fig. 32).

In the more severe infections suppuration may occur with the formation of an abscess cavity, or necrotic areas may appear in various parts of the exudate, and these become converted into pus. Commonly, there is one cavity which results from the conjunction of several pus foci; occasionally the entire pelvis may be riddled with abscesses. Multiple foci of suppuration are commonly of thrombotic origin and really belong to a different class from the simple cellulitic abscess. These large abscesses, dependent upon their proximity to one of the hollow organs, are apt in the course of from twenty to seventy days to point, and unless they are evacuated by operative measures, to break into the rectum, bladder, vagina or on the skin above Poupart's ligament or into the peritoneal cavity; and if the pus is completely evacuated, the cavity closes rapidly. Unfortunately, when these abscesses open spontaneously, it is seldom at the most dependent point or there may be other or more remote foci; hence the pus is not completely evacuated and the septic process may be kept up for weeks or months. Sometimes the abscess does not open, and nature cures the condition by encapsulating the pus. The wall of the abscess is thickened and becomes firm with fibrous tissue, while the more fluid part of the pus is absorbed. Such a tumor may persist in the pelvis for years, gradually shrinking in size. The writer has watched several of such cases over a period of years.

It is important to note that the encapsulated germs do not always lose their virulence, but may, on the occasion of subsequent traumatism or operation, break out with increased virulence and cause a bacteriemia. This was shown in one of the writer's patients, who went through a long postpartum cellulitis, in which an immense exudative mass gradually and slowly absorbed and diminished in size, but never completely disappeared. Three years later she came into the hospital pregnant, and gave birth

spontaneously to a small baby without any vaginal examination being made. The placenta was also expelled spontaneously; she died eighty hours later of a streptococcic bacteriemia, hemolytic streptococci, two hundred colonies to the cubic centimeter were found in the blood. Autopsy showed that a cervical tear had been reopened and the solution of tissue had passed through an encapsulated pus cavity in the left parametrium.

Unless there has been considerable trauma of the soft tissue, it is remarkable to see how little scar tissue is left after these connective tissue abscesses heal. On the other hand, when there have been extensive lacerations and trauma of the soft parts, as tears through the cervix, lower uterine segment, and into the base of the broad ligament, the woman is left with a permanent displacement of the uterus, owing to the contraction of the cicatricial tissue.

The pelvic cellulitis may be complicated by femoral thrombosis and phlegmasia alba dolens, though we are of the belief that the more severe cases should be regarded as a septic thrombosis with an accompanying cellulitis; it is conceivable also to believe that an immense exudate may of itself be sufficient to cause compression of the pelvic veins and produce edema of the leg or thigh. This is so especially, when the exudate is in the anterior portion of the pelvis, between the peritoneum and the pelvic bones.

Chronic pelvic cellulitis is but an extension of chronic inflammation and is usually found to be associated with infected cervical lacerations. It is seen in the posterior uterosacral cellulitis and the lateral parametritis so constant in chronic endocervicitis. We believe that, next to endometritis, cellulitis is the most frequent pathological lesion found in puerperal infection. Seven to eleven per cent of all gynecological cases have a coëxisting parametritis.

Symptoms.—In the mildest form evidences of previous cellular infection are commonly found during gynecological examination in the form of a thickening or scar in one of the fornices, drawing the cervix over to one side. This may have caused no noticeable symptoms during the puerperium or there may have been a slight rise in temperature, and a little local tenderness in one or the other iliac fossa, but of such brief duration as not to have directed the attention of the attendant to the pelvis.

In the severer grades there is usually a history of a traumatic labor with instrumental delivery, or of a dry labor where frequent vaginal examinations have been made or pituitary extract was given to hasten the labor. Then, on the third or fourth day postpartum, the patient com-

plains of pain in one or the other inguinal regions and the temperature rises.

Nearly always the symptoms of endometritis precede those of parametritis, and the early symptoms of the local lesion are frequently masked by a variable degree of toxemia, manifested by pyrexia, acceleration of the pulse rate, headache, anorexia, malaise, etc. It is usually possible to determine the time when the infection passes from within the confines of the uterus and reaches the broad ligament. *A chill or chilliness, febrile reaction, increase in the pulse rate, and the occurrence of marked local pain are constant symptoms;* these, with the general manifestations of the febrile state, such as headache, muscle soreness, anorexia, sleeplessness and restlessness make up the symptom complex. All movements cause pain in the affected side. Nausea and vomiting are generally absent in cellular infections, unless the process involves the overlying peritoneum.

The degree of localization is determined by the degree of virulence of the infecting organism and the local and general resistance of the patient's tissues. *Traumatism and hemorrhage definitely reduce this resistance.*

As a rule, there is a prompt tissue reaction at the seat of infection, demonstrable as an exudate which is poured into the cellular tissues, except in the case of infections from hemolytic bacteria, when there is little or no local reaction, hence less demonstrable lesion.

The symptoms of an acute localized cellulitis may appear three or four days after delivery, or may be delayed as late as the second week. The fever which may be present soon after delivery will either have diminished for a time, only to increase again with the development of the local lesions, or, if there has been little or no fever in the first week, a rising temperature with morning remissions will mark the pouring out of the exudate into the cellular tissue. The local inflammation may be ushered in with rigor, but repeated chills are uncommon in cellular inflammation. The pulse rate rises and falls with the temperature; a *persistently high pulse rate is uncommon in parametritis.*

Pain is present in one or the other lower abdominal quadrant, but it is not such a marked feature as in pelvic peritonitis. It is most severe when the effusion develops rapidly. It is aggravated by movement; often it extends down the thigh of the affected side or up into the loin. In slowly developing exudates pain may be entirely absent.

When the exudate extends into the iliac fossa, the leg on that side is generally drawn up to relax the tension on the psoas and iliacus muscles, as the sheaths and connective tissue bundles of these are involved in the

inflammatory process. Defecation and micturition may be painful when the exudate is contiguous to the rectum or bladder. Diarrhea with the passage of mucus with the stool, and the signs of cystitis, indicates extension of the inflammatory process to the rectal and vesical walls.

In addition to the local discomfort or pain and pyrexia, the general symptoms of cellulitis are those of a toxemia, i.e., sleeplessness, restlessness, anorexia, thirst, headache and constipation. The tongue is coated, but the severe oral changes are generally absent and the general health little affected except in those prolonged cases which go on to suppuration, or where there has been extensive blood loss at the time of delivery. The temperature gradually subsides in the course of a week or ten days, *only to rise again on some pelvic manipulation, which squeezes the toxins out of the limiting exudate.* It must be remembered that an exudate is always a conservative process, which tends to limit bacterial extension.

Physical Signs.—On examination, some evidence of uterine infection is generally found; the uterus may be tender and larger than is justified by the period of the puerperium, the lochia may be profuse, offensive or purulent discharge and lacerations of the cervix or vagina are frequently present.

An exudate, extending from the cervical rent in the base of the broad ligament, is not usually found for some days after the onset of the symptoms. At first the edema and infiltration of the tissues give only a feeling of tenderness and greater resistance in one or both sides of the vaginal vault, but as the effusion becomes more fibrinous in character, a definite, *hard, unyielding mass, running out laterally from the uterus or forward to the anterior pelvic wall, blending intimately with the uterus,* will be recognized. This mass becomes harder and more fixed, causing depression of the vaginal vault on that side or surrounding the cervix like a collar, effacing the portio. The uterus is immobile and either set in this mass of exudate, which blends intimately with it, or is displaced toward the unaffected side.

Recto-abdominal examination will show the pouch of Douglas and the posterior part of the pelvis free from mass, and permit the palpation of the smooth hard posterior surface of the broad ligament. As the mass increases in size, it passes forward to the cellular tissues beneath the reflection of the peritoneum on the anterior abdominal wall, and forms a broad band of induration running parallel to Poupart's ligament, or it may extend upward into the loin and reach the kidney.

Differential Diagnosis of cellular inflammations presents great difficulty; especially is it difficult to differentiate these exudates from the intraperitoneal masses so commonly found as a complication of inflamed

appendages. The clinical history does much toward suggesting the diagnosis.

Primary cellulitis is always a sequel of infected labor or operative procedures, which open into the cellular tissues of the pelvis, hence unless such a history is obtainable, pelvic, exudative masses must have their origin in some infective focus in the uterine appendages.

Severe abdominal pain and tenderness with rigidity of the abdominal wall, especially if accompanied by distention and difficulty in obtaining an action of the bowels, clearly point to a peritoneal infection. Acute pain and abdominal tenderness may generally be taken as indicative of peritoneal involvement. The fever is usually more marked in cellulitis than in peritonitis, while the relation of the pulse rate to the temperature is higher in peritonitis than in cellulitis.

The differentiation between cellulitis and peritonitis by physical signs is very difficult in the acute stage of inflammation, because both the peritoneum and cellular tissues are involved. Frequently a positive diagnosis cannot be made until all of the acute symptoms have subsided.

The distinctive shape of the inflamed uterine appendages is only met with in the chronic swellings in the "cold stage." The principal points in differentiation are:

1. The history.

2. Pelvic cellulitis is more often unilateral, while peritonitis is commonly a bilateral affection.

3. In pelvic cellulitis the mass is at the base of the broad ligament and extends forward toward the anterior pelvic wall and intimately blends with the uterus. In pelvic peritonitis the mass is found behind the broad ligament and in the *cul de sac* of Douglas; the exudate is in the posterior quadrant.

4. The presence of a fluid exudate in the pouch of Douglas which pushes the uterus forward makes a peritoneal infection certain. A hard ring around the rectum, on rectal exploration, suggests a peritoneal rather than a cellular inflammation.

Septic thrombosis of the pelvic veins may occasionally give rise to a tender circumscribed swelling or a thickened cord, extending from the uterus outward toward the pelvic wall. Some exudation into the perivenous cellular tissue always surrounds these thrombosed veins and so adds to the size of the swelling. High fever of a remittent type, with rigors and sweats and other pyemic symptoms, is generally present. The small size of the pelvic mass, with the serious character of the general symptoms, is characteristic of thrombophlebitis.

Prognosis.—Localized cellulitis terminates in resolution or suppura-

tion; the exudative protection is so complete that recovery is the rule, for the surrounding exudate confines the toxins. If the mass becomes harder, smaller and insensitive, and examination or manipulation fails to cause a rise of temperature or increase in the leukocyte count, we may conclude that time will accomplish complete absorption, leaving but one sequela, i.e., pelvic varicosities.

Evening elevation of temperature with remissions or intermissions, increased size and sensitiveness of the mass, and an increased polymorphonuclear percentage, suggests suppuration. The diagnosis is positive when fluctuation can be determined. When the abscess has been opened and good drainage established, the fever subsides and at once the general condition improves. The rapidity with which the toxic symptoms abate is characteristic of an uncomplicated cellulitic abscess. Some displacement and permanent fixation of the pelvic organs will result from cicatrization in cases of extensive suppuration. The most serious consequences are those due to a spread of infection to other structures. Infection of the large venous plexuses in the pelvis, with septic thrombosis and pyemia may occur, but fortunately is rare. The writer has seen but one such case in a very extended experience.

Rupture of the cellulitic abscess into the peritoneal cavity is also very unusual, but rupture of abscesses in the paravesical space does occur into the bladder, giving rise to cystitis, which at times is intractable. Pyelitis and pyelonephritis are possible complications.

CHAPTER V

PUERPERAL INFECTIONS—(Continued)

Infections of the veins—Thrombophlebitis of the femoral or of the saphenous veins—Puerperal pyemia—Treatment of the infections of the femoral and pelvic veins—Radical treatment—Bacteriemia—Avenues of entrance—Prognosis in puerperal infections—Treatment of puerperal infections—Parametritis and perimetritis—Pelvic peritonitis—Operative measures—Infusions of citrated blood—Gas bacillus infection.

INFECTIONS OF THE VEINS

There are two chief types of infection which attack the veins:

(a) Thrombophlebitis of the femoral vein or of the saphenous vein.

(b) Puerperal pyemia.

Thrombophlebitis of the Femoral or Saphenous Vein.—This type of infection is characterized by edematous swelling of one or both lower limbs, without severe constitutional disturbances, except a moderate pyrexia.

In puerperal pyemia, septic emboli are deposited in distant parts of the body and repeated rigors occur, which are followed by marked temperature rise and pulse flights, with grave, frequently fatal, toxemia.

Clinical Course.—Simple thrombosis of the saphenous or femoral veins, or of the iliac, is attended with edema of the lower extremity, beginning at the foot and extending to the trunk. The onset is usually sudden, beginning during the second week of the puerperium, though the occurrence of the pain and local edema is commonly *preceded by tardy involution of the uterus, the persistence of the red lochia,* and a *slight but continuous elevation in the evening temperature.* Coincident with the occurrence of local pain, there is an increase or a recurrence of the temperature. The pain is located in the foot or in the calf or in the groin, and the edema, which begins in the foot, rapidly involves the whole limb. The skin is cool, translucent, and mottled, or marbled with distended veins. The pain and temperature gradually subside in a week or ten days. The left limb is more commonly affected than the right, but in one third of the cases both limbs are involved.

Beside the general edema of the limb, the glazed stretched appearance of the skin, and the marked enlargement of the superficial veins, there may be fullness, induration, and tenderness at some one point

along the course of the involved vein. This focus is most commonly *found in the groin* or *over the calf of the leg* below the *popliteal junction.*

The writer believes that these thrombophlebitic lesions are always the result of infection. This view is concurred in by DeLee, McLean, and others. No case has ever been observed in which there has not been some elevation of temperature before the occurrence of the local lesion.

Symptoms.—Many cases of sudden death, during the puerperium, from embolism, are preceded by mild febrile symptoms, rapid pulse, or local symptoms which indicate that infective disease of some kind exists in the pelvis. With the exception of these rare accidents, the disease as a rule runs a favorable course, the fever keeps a low level, the pain disappears, and then gradually the swelling subsides, the general health, appetite and sleep are but little disturbed, and recovery takes place in from two to six weeks. The edema, however, persists for some time, especially the edema of the leg and foot, which may appear toward the end of the day, for months.

A more serious condition is *thrombophlebitis of the pelvic and crural veins, which is but an outward extension of the infection of the uterine veins,* part of a general pyemic process. In this form of thrombophlebitis, the pain and swelling of the leg are preceded by the signs of endometritis and pelvic inflammation, or the original intrapelvic symptoms may have been overlooked or gone unnoticed until extension to the crural veins has occurred. There may be an initial chill, which is followed immediately by thrombotic localization in the limb; this occurs usually after the eighth day, or as late as the twentieth. The woman experiences pain in the groin or in the calf of the leg. The swelling is first noticed in Scarpa's triangle and the labium. The groin is commonly the site of greatest pain, and the thrombosed femoral may be palpated as a swollen sensitive cord, due to the associated periphlebitis, as it passes under Poupart's ligament. This is easily demonstrated in thin women. The upper thigh is immensely swollen and has a white opalescent or somewhat yellowish tint; is hot and sensitive; frequently the tension is so great that the tissues will not pit on pressure. Within a few hours, and always within a day or two, the whole limb is involved; the leg and foot become edematous, but not infiltrated. The constitutional symptoms may or may not be ushered in with a chill, but fever is always present and the patient shows signs of a severe illness, as of a pyemic process.

Both limbs may be involved one after another, for this type of phlebitis is but an extension of a septic thrombosis of the pelvic veins,

which has crept out through the pampiniform plexus to the obturator or hypogastric veins, thence to the iliac, and then to the femoral. There is always a periphlebitis, which adds to the tissue block and thus increases the areas of infiltration. The fever is irregular, with remissions, but does not intermit. The pulse is high and the course of the disease protracted. It may be weeks or even months before the infiltration of the tissues disappears and the venous obstruction subsides. The thrombosis may even extend to the arteries and terminal gangrene result.

Commonly an inflammation or stasis of the lymphatic channels accompanies the inflammation of the veins, for the process has an intrapelvic origin; the infection spreads out of the pelvis and down the leg, so that the surrounding cellular tissue is solid, with infiltrated lymph.

Every *pelvic phlebitis is attended with more or less cellular inflammation* and may, because of the proximity of the peritoneum, excite a local peritonitis; consequently the cellular and peritonitic inflammation may mask the true lesion in the vein.

Puerperal Pyemia.—This form of puerperal infection is far more common than is generally recognized. Lea, from his extensive post-mortem studies, claims that it is responsible for from thirty to fifty per cent of all deaths from infection.

In *nearly all cases intra-uterine manipulations have preceded its onset*, i.e., careless treatment of placenta previa, or postpartum hemorrhage, or a curettage for uterine infection has been recently done.

The writer has recently seen three cases of direct infection of the placental site, following long dry labors, without any intra-uterine manipulation, except *frequent vaginal* examinations. This confirms the observations of Slemons, that long dry labor reduces the endothelial resistance of the amniotic cells, and allows infective bacteria to penetrate the placenta and enter the placental site; hence all cases in which there is an interpartum temperature should be viewed with suspicion.

Clinically the condition is characterized by repeated rigors, with sharp elevations and marked remissions of temperature. The rise of temperature immediately follows the rigor, and this is succeeded by a sudden remission and profuse sweat. Anemia and wasting rapidly develop, owing to the rapid destruction of the cellular elements of the blood. The pulse rises with the temperature, but has not the proportionate fall. The prostration is extreme, though the patient may feel fairly well and be able to take food in the intervals between rigors. The red cell destruction is rapid, the hemoglobin falling 10-50 points in a period of a few days. There is little or no leukocytosis. Emboli may locate in

the lung, pleura, endocardium, and brain. *The physical signs are not definite, nor are they usually marked.* Involution may be arrested, but the abdomen is generally negative, unless the condition is complicated by parametrial or peritoneal infections as well. *Commonly there is a history of retarded involution;* the uterus is relaxed, the lochia profuse, there are painful uterine contractions, and a slight evening temperature before the extension into the veins occurs. Occasionally there is a slight tenderness on deep pressure over the course of the ovarian or external iliac veins.

Vaginal examination may reveal by careful palpation a small slightly tender swelling, either high up in the ovarian region, due to phlebitis of the ovarian plexus of veins, or at the base of the broad ligament, when the uterine veins are thrombosed. Williams claims that the thrombosed veins may be palpated as tender "worm-like" masses in the broad ligament, by recto-abdominal touch. An extensive clinical experience convinces us that the *diagnosis must be made from the clinical symptoms, not by physical signs;* as the tactile sense is seldom able to differentiate the thrombosed veins from coincident parametric and peritoneal lesions. Signs of ulcerative endocarditis may be made out and appear relatively early. About half of the fatal cases show this complication. Septic embolic pneumonia of the patchy type is an almost constant postmortem finding, though it is seldom detected during life.

The prognosis is always grave; death occurs in from one to three weeks from general asthenia and toxemia, though the patient's life may be suddenly cut short by sudden death from pulmonary embolism. Less than twenty per cent spontaneously recover. Beach reports a case of recovery after eighty-six days in a woman who had repeated small transfusions. Beck and the writer have seen four recoveries in seven typical cases, as a result of the expectant plan of treatment, in which fresh air and small repeated blood transfusions were the only therapeutic measures used.

Those patients in whom there are embolic abscesses in the superficial tissues, seem to develop the greatest resistance. One of the author's cases, which recovered after 109 days of fever, developed abscesses in both arms, both legs, and in the lungs; but throughout her leukocyte count never fell below 16,000.

Treatment of Infections of the Femoral and Pelvic Veins.—In the management of thrombophlebitis of the femoral or saphenous veins the treatment is largely symptomatic, for, until a collateral circulation is established, swelling of the limb must necessarily persist. As has already been stated in discussing the pathology, when the femoral vein is involved, there is usually a periphlebitis in the cellular tissues of

Scarpa's triangle. This effectually blocks the deep return circulation from the limb, consequently the foot, leg and thigh become enormously swollen and the patient complains of intense stretching pain in the thigh, due to the extreme tissue tension.

Proper elevation of the limb, to favor the return circulation, is the basic fundamental in the treatment. Heat and elastic support contribute somewhat to the patient's comfort. Our plan has been to elevate the leg and thigh, *having the leg slightly flexed on pillows, the limb is then wrapped from the toes to the groin* in cotton batting, which is held in place by a firmly applied stockinet bandage; this gives elastic support and yet sufficient pressure, to favor the establishment of a return circulation. *Codein or morphin in small and repeated doses must be used for the relief of the pain.* In addition to this it may be necessary to relieve the pain due to the cellulitis in the groin by the application of cold; this is done by placing an ice bag on the groin over the femoral.

The constitutional symptoms, which are always pronounced during the active thrombus formation and the invasion of the thrombus with cocci, are treated by general supportive measures, i.e., the copious ingestion of water, evacuation of the bowels by daily enemata, and stimulation of the leukocytic reaction by the administration of leukocytic extracts. The pyrexia needs no medical treatment, as it is but an index of the tissue reaction, which is attempting to localize and check further bacterial extension.

After a week or ten days the remissions in temperature become more marked and the swelling and edema in the leg gradually tend to subside. *The temperature should be normal for at least ten days before the patient is allowed to use the leg,* and she should never be permitted to get up and about without proper elastic support, as the swelling in the terminal portion of the limb will persist for months after a collateral circulation has been established; for the collateral circulation is made by gradual enlargement of the superficial veins, which have not the natural tissue support of the deeper vessels.

Occasionally the tissues involved in the periphlebitis in Scarpa's triangle, in the popliteal space, or the calf of the leg, may suppurate. Should this occur, it is well to allow the abscess to point before it is incised, otherwise we are apt to have an indolent ulcer result, which has little or no tendency to heal, owing to the poorly nourished surrounding tissues.

When the diagnosis of a thrombophlebitis of the pelvic veins has been made, two plans of treatment have been advocated, (1) the expectant and (2) the radical. We believe that *thrombophlebitis is a con-*

servative process, an evidence of *nature's effort to localize the infective organism in the veins by the formation of a blood clot to block its path.* Bacteria liquefy the thrombus and then advance through it. When again there is a reaction on the part of the blood to this advance, more clot is formed and another temporary block is established. As this is the fact, it is easily appreciated that, whether the bacterium advances or remains localized, will depend on the character of the bacterium, its virulency, and the resistance of the patient's blood stream. *The chill is the index and the evidence of its advance; the fever is but the symbol of tissue reaction; the intermission in the fever, the sign of the temporary check.* If the leukocytic resistance and the fibrin forming elements of the blood are sufficient, the lumen of the vessel becomes obliterated with an organized clot and the temperature gradually subsides. Upon the other hand, if the cellular elements of the blood are deficient, or the bacterium is hemolytic, the infecting organism is bound to pass through the fibrinous obstruction and reach remote points by the blood stream.

Unfortunately, the *leukocytic resistance is always low in thrombophlebitis,* the white cell count will seldom be found to be more than ten thousand, and the red cells and hemoglobin are progressively diminished; hence, in the expectant treatment, knowing what is necessary for nature to do in order to control the advance of the infection, we can simply aid it by general supportive treatment.

It has been our practice to treat these patients with fresh air and sunshine, having their beds removed to the roof, and, if possible, having them sleep in the air. This materially improves their appetite and digestion, so, during their remissions in temperature, they are able to take food and digest it. The kidney elimination is stimulated by the copious ingestion of water and by enteroclysis, using a glucose and bicarbonate of soda solution, and, when the temperature rises, their resistance is maintained by the use of lactose in fruit juices.

In addition to this plan of general sustaining treatment, we have found that *repeated small transfusions of blood* (300-350 c.cm. at a time) *gradually increase the cellular elements in the blood stream* and thus offer a better tissue reaction to the bacterial invasion of the clot. These transfusions are given of citrated blood at three or four day intervals. A full blood count is taken before and after each transfusion and, not until the red cells and hemoglobin show a definite improvement, and are able to maintain this improvement during the periods between the transfusions, are the blood injections omitted. If the patient is to have

a favorable outcome, it soon becomes apparent that the leukocyte increase is maintained and the red cells rapidly increase.

Radical Treatment.—The radical treatment consists of ligation and excision of the infected ovarian and hypogastric veins. This was first done by Freund in 1897. In 1902 Trendelenburg reported five operations with an improved technic, demonstrating the advantage of his posture in exposing the field of operation; and recently Miller, of New Orleans, has published an extensive article on the status of ligation in thrombophlebitis. His review included all the reported cases to 1917, with a corrected mortality from ligation of 32.9 per cent.

Unfortunately, the diagnosis as to the exact location of the thrombosed vein is difficult to make clinically. Williams claims that, by manual palpation, the thrombosed veins may be recognized as hard, sensitive cords in the base of the broad ligament; this has not, however, been our experience, for it is usual for the ovarian vein, rather than the uterine, to be the seat of the thrombosis, and hence bimanual palpation will commonly fail to locate the thrombosed veins, and when masses have been palpated in the base of the broad ligament, operation or autopsy has usually shown them to be of a parametritic character. The diagnosis, however, is easily made on the history and clinical picture and low leukocyte count.

It would seem from Miller's analysis that, if the ovarian veins alone are involved, the outlook for operation is favorable, if done at the proper moment. When both the ovarian and the hypogastrics are involved, the outlook is hopeless. We are not at all sure that ligation will show better results than transfusion, for the best results of the advocates of ligation have been attained in the subacute and chronic cases, which, in our experience, are the cases which get well spontaneously. Again, it has been our experience that there is more or less perivascular inflammation in the cellular structures surrounding the veins, as well as some complicating peritonitis. *If the operation is to be a success, it must be done in the case which is free from lymphatic or peritoneal infection.* Hysterectomy defeats the purpose of confining the bacterial advance to the thrombus already formed. This statement is supported theoretically and by the clinical experience of most observers.

Technic.—The technic is relatively simple to the experienced abdominal operator. With the patient in a high Trendelenburg posture, the abdomen is opened by a long median incision, the pelvis packed off from the general cavity, and the sigmoid lifted toward the median line. The parietal peritoneum is now divided to the outer side of the sigmoid, and, with blunt dissection, the pelvic veins may be easily and completely ex-

posed. The thrombosed vein is isolated, ligated above and below the thrombus, and the vein severed. It is not necessary to excise the intervening portion. A similar procedure is done on the opposite side. In our cases we have ligated both internal iliacs close to their junction with the common, as well as both ovarians. By this extensive exclusion the best results have been obtained. The reflected peritoneum is then brought into place and sutured and the abdomen closed without drainage. *The fundamental principle is to disturb the pelvis as little as possible by manipulation.*

Bacteriemia.—Bacteriemia means the presence of bacteria in the blood. It is an acute infectious disease, produced most frequently by the streptococcus pyogenes, or the staphylococcus. These cocci with their toxins, produce changes in the blood, destroying the red cells as well as the leukocytes, and cause degenerative changes in the organs through which they pass, notably, the heart, the liver and the kidneys.

Besides the streptococcus and the staphylococcus, which are the most common invaders of the blood stream, the pneumococcus, the bacillus pyocyaneus, the gonococcus, the bacillus aërogenes capsulatus, and several anaërobic bacilli have been found in blood cultures.

Avenues of Entrance.—In postabortal and puerperal infections entrance is gained into the blood stream by two routes: first, by lymphatic extension, second, by direct invasion of the venous radicles and sinuses. Each mode of invasion proceeds in a definite manner, and the clinical pictures produced differ so much that it is generally possible to make a differential diagnosis. Occasionally, however, the pictures are indistinct and differentiation is impossible.

The lymphatic form develops from an endometritis, the infection in turn extending to the myometrium and the para-uterine lymphatics; but it is so virulent that, instead of exciting an active reaction in the parametrium and para-uterine lymph spaces, the reaction is simply a serous exudate with local edema and the infection proceeds directly into the blood stream or through the lymphatics to the peritoneum, exciting an acute, purulent peritonitis.

On the other hand, the vascular form almost invariably begins as a uterine phlebitis, primarily as an infection of the thrombi in the placental site, with an extension of the infected thrombi into the veins. From these infected thrombi the bacteria enter and multiply in the blood, and subsequently locate in distant organs, such as the pleura, the lung, the endocardium, and the brain. Occasionally the thrombi may suppurate, but this is not common in streptococcemia. However, as a result of such suppuration, bits of infected fibrin or actual pus may get loose, and

be carried away by the blood stream to remote parts of the body, and there locate and cause local abscesses; the lung, kidneys, and the brain are the points most frequently reached by these infected emboli.

In blood stream infections the local pathological reaction is inconsiderable; consequently the local symptomatology is insignificant, for, whether the bacteria enter the blood stream via the lymphatics, or via the veins, their transit is so rapid, and the tissue reaction caused so insignificant, that appreciable local lesions must necessarily be absent.

For the entrance of bacteria into the blood stream there must be a puerperal wound, which is inoculated by bacteria. This may be at any point in the genital tract, the vulva, vagina, the cervix, or in the placental site. DeLee states that even a tear of the frenulum may afford access, through which a virulent streptococcus may reach the blood stream. Here, as in other puerperal lesions, the activity of the process depends on the virulence of the coccus and the resistance of the patient. Women who have had severe postpartum hemorrhage, or have been toxic, prior to their delivery, offer less resistance to coccal invasion than women whose antepartum or interpartum period has been less depleting.

In the lymphatic varieties of bacteriemia the findings at autopsy are constant and typical. (1) Endometritis gangrenosa or metritis desiccans (Garrigues), in which the whole tract is covered with a grayish sloughing diphtheroid exudate. (2) Parametritis and lymphangitis, the lymph vessels at the sides of the uterus being filled with purulent fluid which exudes from the cut surface. The connective tissues may be infiltrated and edematous, and this cellulitis may spread so fast and so far as to justify the name which Virchow gave it, "Erysipelas puerperalis malignum internum." The changes in the parametria may be simply a serous, infectious edema, or necrosis may occur, and a real phlegmon result. (3) Pelvic peritonitis, then general peritonitis. (4) If the patient lasts long enough, pleuritis and pericarditis. (5) Gastritis, enteritis, and colitis. (6) With the general pathology of acute infectious disease, i.e., swollen spleen, fatty degeneration and cloudy swelling of the muscle fibers, especially of the heart, and cloudy swelling of the liver and kidneys. Bacteria in immense numbers are found in the minute capillaries of these organs. (7) The findings of metastases in the lungs, heart, brain, joints, and connective tissues.

In the vascular forms of bacteriemia the lymphatics are not involved at all, or if so, to a very decidedly less extent. The veins of the placental site are filled with large thrombi, which are swarming with bacteria. The bacteria erode the endothelial lining of the vessel, fibrin is therefore deposited on the eroded surface, and a clot occludes the lumen; and

this process advances through the venous plexuses of the broad ligament into the ovarian and iliac veins, even to the vena cava.

From the surface of these thrombi, bacteria are liberated into the blood stream, and if they are strong enough to multiply in it, a fatal bacteriemia may result. If the bacteria are less virulent, the process becomes more chronic, the thrombi undergo puriform softening, and solid bits of thrombus or droplets of pus break loose and, floating in the blood stream, lodge in distant parts of the body, setting up new foci of suppuration, which is actually a condition of pyemia.

Symptoms.—A period of incubation of from one to three days usually precedes the outbreak of the severe symptoms. Occasionally threatening prodromata appear within a short time after the inoculation, and the woman becomes seriously sick and may die within thirty-six hours. Ordinarily the prodromal stage is manifested by the signs and symptoms of the local process, from the site of which the bacterial invasion of the blood has extended.

In the consideration of the pathology, we have shown how bacteria may enter the blood stream from a local ulcer, through the lymphatics, or from an endometritis or parametritis, or by way of the placental site with infection of its thrombi. It is, however, frequently impossible to determine when or how the germs get into the blood. Since our bacteriologists have been using anaërobic methods, we have frequently been able to cultivate the streptococcus from the blood where the diagnosis of a purely local lesion has previously been made.

The following syndrome is indicative of a serious bacteriemic infection, though it is claimed that absorption of toxins in large amounts, will produce similar symptoms; this I cannot verify from personal experience. Blood invasion is ushered in by a severe chill, lasting from five to thirty minutes; during the chill the skin is pale, the face is pinched and the lips and fingers cyanotic; the temperature rises rapidly to 103-105° F. and the pulse increases at once above 120, varying from 130 to 160. At first the pulse is full and bounding, but it soon becomes soft and compressible, for the toxins in the blood weaken the heart muscle. Owing to the rapid destruction of the red blood corpuscles, the oxygen carrying power of the blood is diminished, and the patient exhibits marked pallor, the finger tips are cyanotic and the respirations are hurried, and the woman looks profoundly sick. The white blood cells show no tendency to increase, owing to the intense and overwhelming intoxication. Owing to the rapid production of toxins, the non-striated muscle in the heart and intestinal tract becomes toneless, due to cloudy swelling, and as the heart weakens, the blood pressure falls and there is more and

more tympany from intestinal paresis. This further embarrasses the heart and respiration.

Malaise becomes a prominent factor early in the attack, the woman appears prostrated and apprehensive of impending danger. Headache and sleeplessness are constantly complained of and even though the patient has no pain, she does not sleep. This symptom is particularly ominous. The mind may remain clear until near the end; this however is unusual, as a mild delirium is the rule. As the endocarditis develops the delirium becomes more marked.

The bacteriemic symptoms may occur alone, or be succeeded by the symptoms and signs of a purulent peritonitis, i.e., nausea, vomiting, and pain. These, with the facies hippocratica, show the end is not far distant.

If the bacteriemia has occurred as the result of rapid lymphatic invasion from a coccal endometritis, local pelvic symptoms may coexist. The lochia are usually profuse and putrid, the result of a gangrenous endometritis, though in the severer types the lochia may be scant and free from odor. The odor is pungent and the puerperal wounds become necrotic. Signs of peritonitis, such as tenderness, tympany, spreading rigidity, ileus, etc., begin, and if the patient lives long enough the picture becomes one of virulent peritonitis. When this occurs the *temperature may go down, but the pulse always rises and the tongue becomes dry.* A peculiar sickening, fruity odor is noticed about the patient and while the patient feels easier, the objective symptoms grow worse. The body is cold, the face flushed, and beads of cold perspiration stand out on the forehead, while the nose, lips and ears are of a leaden gray. Death usually occurs in coma preceded by pulmonary edema.

The disease lasts for from two to ten days and is especially virulent, if it begins during labor, when the course is usually short and violent. Eruptions on the skin resembling scarlatina and measles occasionally occur. This has nothing in common with true scarlatina, though the pregnant woman is not immune from the disease. It is really a toxic streptococcic erythema. There is no angina and this helps in making the differentiation.

The Prognosis in Puerperal Infection.—The estimated death rate from postpartum septic infections varies from five to thirty per cent. Unfortunately, it is difficult to obtain reliable statistics, owing to the fact that many of the deaths are reported as pneumonia, pulmonary embolus, endocarditis, and so forth. It is true that the chief causes of death, judging from autopsy findings, are septic foci in the lungs, malignant endocarditis, pulmonary embolism, and septic peritonitis but these condi-

tions are secondary to the primary septic focus in the pelvis. Occasionally, in cases of severe infection, no gross lesions can be found at autopsy and it must be assumed that the death is due to the severity of the toxinemia or bacteriemia.

Postabortal infections are less liable to be attended by fatality, for tubal and pelvic peritoneal lesions, rather than lymphatic and blood stream infections, commonly follow upon an infected abortion, and furthermore, because the uterus is well within the pelvic cavity, isolation of the infection by peritoneal adhesion is more likely.

The prognosis in the particular case depends (1) on the site and extent of the lesion, (2) on the form and virulence of the organism, and finally (3) upon the resistance of the patient.

Broadly speaking, vulvovaginal infections, uncomplicated endometritis putrida, cellulitis, femoral thrombosis, and pelvic peritonitis give the most favorable prognosis, while it must be admitted that excessive blood loss during or following labor materially reduces the patient's resistance and contributes to the severity of any of these lesions. Fatal results from any of the foregoing are unusual.

Vulvovaginal infections, although sometimes attended by high temperature and copious discharge, usually terminate in complete recovery. The remaining scars seldom cause more severe trouble than to limit the mobility of the contiguous organs and occasion dyspareunia.

Putrid endometritis usually makes a rapid recovery after proper uterine drainage has been established; occasionally, however, extension of the septic process may take place through the placental site or through the lymphatics and a pelvic thrombophlebitis or bacteriemia develop. It is better therefore, in this condition, to give a guarded prognosis.

Cellulitis generally terminates in recovery with or without suppuration. The course of the disease is often prolonged, but if the process is entirely confined to the cellular tissue, the possibility of future pregnancy is not impaired; on the other hand a pelvic peritonitis, which also runs a long course, commonly ends in resolution or a pelvic abscess; in either case the tubes are impaired by the results of peritubal inflammation, and sterility and dysmenorrhea are common sequelae.

In femoral thrombosis (thrombophlebitis), while the prognosis is usually favorable, the possible danger of pulmonary embolism must be constantly borne in mind. Embolism occurs in from five to ten per cent of cases of thrombosis and ends in death more often than in recovery. It happens most frequently in the first ten days after the thrombosis appears, but the danger of embolism is not passed until the temperature has remained normal for at least three weeks. Femoral thrombosis may ter-

minate in the pyemic or more fatal form of venous infection. Marked pyrexia, signs of suppuration, and accompanying thrombosis of the pelvic veins are unfavorable features. Varicosities of the extremity are a constant reminder of the original pathology.

In the bacteriemic form of puerperal septic infection the prognosis should always be guarded. Early onset after labor, severe general symptoms, and an ascending or persistently rapid pulse indicate a severe type of the disease. The degree of pyrexia is not such a valuable index to the virulence of the infection as are the pulse rate and the blood pressure, but generally speaking, a persistent pyrexia is always serious for this impairs the woman's resistance. About 60 per cent of the severer types of bacteriemia end fatally, usually from toxemia or general peritonitis. The milder types, which are commoner, usually recover; however, a mild type may develop into the virulent type.

Pyemia has a high mortality. Bumm states that the mortality is 83 per cent. Unfortunately we have no accurate way of estimating the cases. According to Miller, this mortality is too high.

The Treatment of Puerperal Infections.—Broadly speaking, we may divide the treatment of puerperal infections into the *prophylactic and curative.* Preventive measures play such an important rôle and so much can be done during pregnancy and labor to prevent the occurrence of infection, that a more detailed discussion of the prophylactic measures seems justifiable.

Admitting that all puerperal infection is the result of inoculation of the puerperal wound with *pathogenic bacteria,* and that while many of these organisms have their habitat in the genital tract, and may migrate into the wounds of the vulva, vagina, cervix, or uterus, clinically we know that *they are most commonly carried into these wounds by the hands or instruments of the accoucheur.* Naturally, therefore, prophylactic treatment must include the proper preparation of the vulvar orifice, sterilization of hands and instruments, and the education of the pregnant woman in local cleanliness and marital abstinence in the later weeks of pregnancy; for it must be realized that the uterine cavity and its contents are sterile at the time of labor or abortion, if such labor or abortion is spontaneous, until it is contaminated with the bacterial flora from the outside. Generally speaking, all local infections such as furuncles about the vulvar, bartholinitis, vulvitis, and so forth, must be cured before it is safe to conduct a delivery.

Credé claims that the underlying principles in the conduct of an aseptic delivery are (1) *to limit as far as possible the puerperal wounds and* (2) *to prevent infection of the necessary puerperal wounds;* hence all

cases should have the *vulva properly prepared before any vaginal examination, and no vulva is properly prepared unless the vulvar hair is removed.*

The number of internal examinations should be limited. In our clinic *we have found that infection is directly proportionate to the number of vaginal examinations.* It is entirely possible to conduct the great majority of labors by *abdominal palpation* and auscultation, by the use of which one can follow the descent and rotation of the presenting part, and from the knowledge thus gained, if used in conjunction with repeated rectal examinations, one may obtain accurate information of the rate of progress and degree of cervical dilatation. *Only in case of accident, such as prolapse of the cord* or *hemorrhage* (placenta previa), or when the progress of labor has been arrested, is it necessary to make vaginal examinations. *When such an internal examination is necessary, the same aseptic care should be observed as is practiced in entering the abdominal cavity.* Preservation of the bag of waters is a great safeguard against infection, particularly infection of the placental site, for Slemons has shown that it is possible in dry labors for bacteria to pass from the interior of the fetal sac directly through the placenta and produce thrombophlebitis in the placental site. Attempts to hurry labor by the introduction of bags, as well as manual dilatation of the cervix or vaginal outlet, tend to increase the possibility of infection. Forceps operations and operative interference generally subject the woman to increased trauma and consequent liability to infections.

In our clinic we prevent perineal and vaginal tears by a median discission of the perineum; this is done just before crowning is complete. In this way we substitute an incised wound through the median raphé for a lacerated wound, which allows the levatores to retract, and this in turn relieves the pressure on the stretched fascial sheets of the vagina, and thus avoids extensive vaginal tears. These median epistomy incisions are readily repaired and, owing to the slight trauma to the tissues, heal kindly.

The physiological conduct of the third stage is also a great safeguard against infection, for by allowing sufficient time to elapse after delivery for the spontaneous separation and expulsion of the placenta, there is little likelihood of fragments being retained. Manual removal and forced efforts at Credé bruise the uterus and diminish its resistance, thus increasing the susceptibility to infection.

Curative Treatment.—The curative treatment is based on the proper recognition of the natural pathology, which must be given its place, for if we admit, and we must, that the *interior of the uterus is a large wound*

surface and is the principal port of entry for bacterial invasion, and that the interior of the uterus if left to itself, undisturbed by interference or trauma, is, except in the presence of the most virulent bacteria, *competent to defend itself against the invading organisms,* one can readily see the fruitlessness and fallacy of intra-uterine manipulation, whether it be by curettage or irrigation.

In our clinic we have not been slow to take up and try out each of the many successive suggestions which have been made for the treatment of primary puerperal endometritis, whether the infection was of the putrid or coccal type. The amount of material at our disposal, in the obstetric and gynecological services of the Long Island College, Methodist, and Jewish Hospitals, has afforded ample opportunity to convince us that *uterine drainage* is the chief contributing factor in the normal uterine reaction against bacterial invasion. Our present treatment, therefore, is along physiological lines, aiding and stimulating nature's own methods of combating the disease.

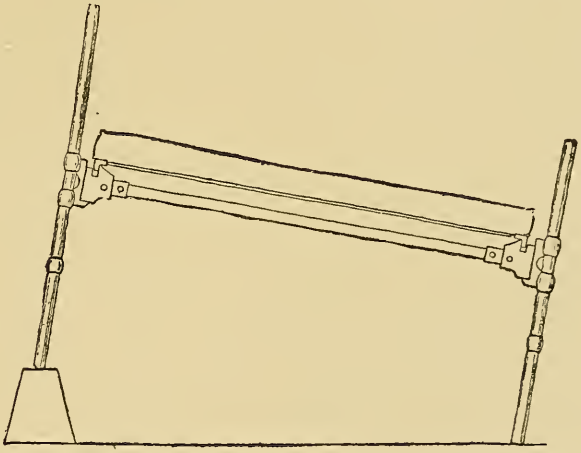

FIG. 33.—FOWLER POSITION BY BLOCKS UNDER HEAD OF BED.

This can be done by (1) postural drainage of the uterus and by (2) securing proper uterine retraction. Notwithstanding my many unconvinced colleagues, *the Fowler elevated trunk posture,* when properly used and supplemented by emptying the vagina from time to time, by having the patient turn over and lie upon her abdomen, the stimulation of uterine contraction and retraction by the use of the ice bag over the fundus, and the administration of pituitrin and the ergot preparations, *does attain this better than any form of intra-uterine irrigation.* Even when we are certain that there is some foreign material retained within the uterine cavity, *the uterus is not invaded,* for the protective granulation zone offers the necessary protection against further invasion.

Only when repaired pelvic structures show evidence of bacterial inoculation are any active measures employed; in such a case, the sutures are removed and the gaping wounds swabbed with the tincture of iodin.

Removal of the sutures usually affords all the drainage the wounds need.

In order to give the reader a more systematic review of the methods employed and the reasons for their employment, we will consider the treatment under the headings of the

1. Local,
2. General,
3. Specific, and
4. Surgical measures.

(1) *Local Measures.*—Theoretically it would seem that it was rational to remove clots, decidua, membranes, and placental fragments, which serve as a pabulum for bacteria, and to neutralize their toxins by mechanical cleansing; certainly this is a surgical principle. *Unfortunately, any attempt to carry it out defeats nature's physiological defense and spreads the disease beyond the limits of the uterus.*

Among the curative measures which have been suggested and used in the past, a few may be mentioned because of their historical interest. (1) *Intra-uterine douches:* almost every chemical antiseptic or proteid solvent has had its advocate, the list will include bichlorid and biniodid of mercury, carbolic acid, formalin, lysol, creolin, alcohol and iodin, saline, and Dakin's solution.

DeLee has summarized the objections to the uterine douche. 1, It is inefficient, for the bacteria are beyond its reach within fifteen minutes after they are inoculated. 2, It is painful, sometimes violent uterine action being set up. 3, If, as sometimes happens, part of the liquid escapes through the tubes into the peritoneal cavity (Kosmak), syncope, vomiting, and peritonitis may result. 4, The nervous shock sometimes causes syncope, even convulsions and coma. 5, The antiseptic employed may be directly poisonous. Over 50 cases of bichloride and as many more of carbolic acid poisoning are on record, the chemical being absorbed by the uterus, or gaining entrance to the blood through the sinuses. 6, Air embolism may occur. 7, Trauma or perforation of the uterus is a possibility. 8, Profuse hemorrhage may result from the manipulation. 9, Chill and fever, the infection having been reinoculated by the douche. 10, Sudden death, which is usually from air embolism, but may be due to cardiac paralysis. 11, Finally, the infection may be carried up higher in the parturient canal, heretofore unaffected.

(2) *Swabbing out the uterus,* with gauze wrapped about a pair of uterine dressing forceps, was a method of cleansing advocated by the opponents of the curet. The gauze was usually wet with carbolic acid,

alcohol, or iodine. *Aside from producing trauma and breaking down the granulation zone, little clinical value can be claimed for it.*

(3) *Curage* or digital removal of the uterine content was very generally endorsed, and some authorities, as Williams, Hirst, and E. P. Davis, advocate aseptic douches before and after the operation. Davis still packs the uterus with washed iodoform gauze.

Our experience has been that all such manipulation disturbs the granulation wall and is followed by constitutional reaction, which takes a day or two to subside.

Only in the presence of dead material or a dead fetus, where the bacillus aërogenes capsulatus has been demonstrated, should any active intervention be advised.

(4) *Curettage.*—Through the teachings of the gynecological surgeon the curet has had very extensive use, though it is *never indicated in the puerperal uterus. The dangers of the curet in the puerperal uterus are those of curettage intensified. It breaks through the wall of leukocytes* and *spreads the infection, allowing the bacteria free access to the lymph channels, and the venous radicles in the uterine wall.* Repeatedly we have demonstrated to our classes the *impossibility of removing the uterine content with the curet,* by taking a uterus which has just been removed by hysterectomy, and systematically curetting its cavity, then opening the uterus and showing the effect of the curettage. The endometrium is removed in streaks and *more is retained than has been removed.* If this is the fact in a firm uterus and when great pains are taken to be thorough, and it cannot be controverted, how much more ineffectual must it be in the soft puerperal uterus. Furthermore, the *bacteria have passed beyond the reach of the curet and invaded the deeper tissues.* Perforation, hemorrhage and air embolism are actual possibilities when the curet is used. For several years we have avoided the use of this dangerous instrument. DeLee aptly puts it, when he says *it seems just as rational to curet the nose and throat in cases of diphtheria, as to curet the uterus in sepsis.*

Numerous *placental forceps* have been devised to remove the uterine content in sepsis. All of them are dangerous, as perforation is no infrequent sequel to their use. Few realize how easily the puerperal uterus can be perforated.

As our experience in sepsis has increased, we have inclined more and more toward the absolute prohibition of every member of our staff from entering the uterus with finger, curet or forceps, and our mortality and morbidity have proportionately decreased. Even the packing of the uterus with iodine soaked gauze has been abandoned. *In a few cases of*

putrid endometritis, with poor drainage and consequent lochiometra, we have followed the suggestion of Ill and introduced into the uterus, to the fundus, a soft, small sized rubber rectal tube, with a strip of packing gauze carried up on either side and loosely packed into the cavity and cervix. When these are in place 50 per cent alcohol is poured through the tube every four hours, leaving the tube and gauze in situ. Ill claims that the presence of the gauze excites contraction and retraction, and the alcohol moistened gauze sterilizes the cavity by actually destroying the bacteria.

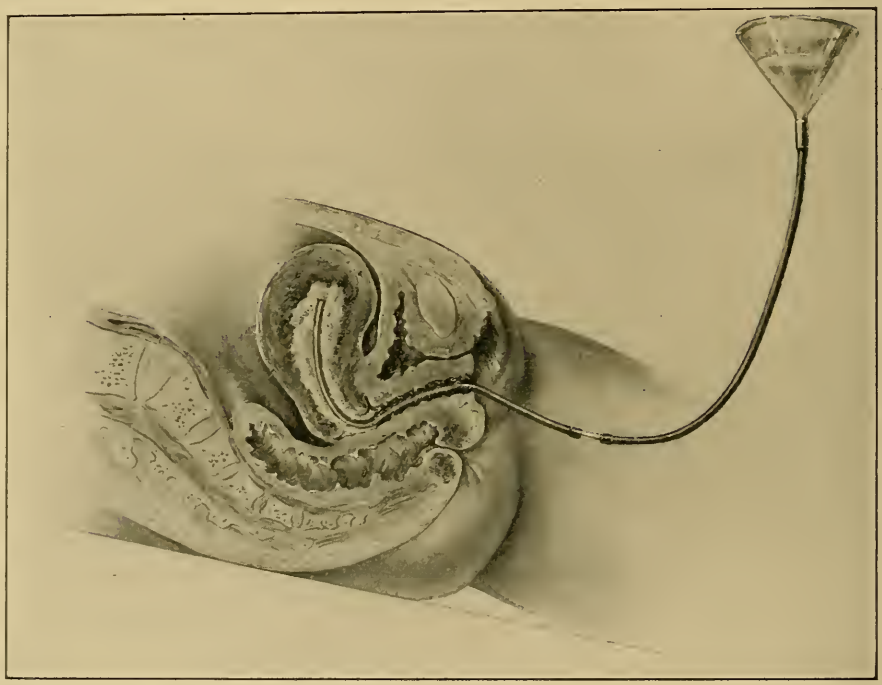

Fig. 34.—Ill's Method of Treating Putrid Endometritis with Alcohol Irrigations.

After the bacteria have passed out of the uterus into or through the myometrium, and have entered the lymphatics or blood vessels, it is evident that no form of local treatment, within the cavity of the uterus, can have the slightest effect on these infective bacteria, except to push them further along and disseminate their toxins into the general circulation. We have already shown, in the pathology, that *lymphatic extension either ends in a parametric exudate, which is definitely conservative and protective, or in a pelvic peritonitis,* which, if confined to the pelvis by proper treatment, is just as conservative a process as the tissue reaction in the parametrium; or the bacteria may pass into the blood and produce a bacteriemia, which is beyond the reach of local treatment.

In parametric invasions our plan of management has been entirely expectant in the belief that the reaction in the cellular tissues is fundamentally protective, an attempt to confine the bacteria within a mass of exudate; hence, this is favored by body and tissue rest. The *patient is placed in the Fowler posture* and *pelvic structures are not handled; the pain is relieved with opium,* in the form of morphin or codein hypodermatically or by rectum, *and the application of an ice bag* over the affected part. *Catharsis is avoided,* and the lower bowel kept empty with small low enemata. *Blood counts are made daily,* for the leukocyte count shows the degree of tissue resistance. A high white cell count usually precedes the drop in temperature. Only about six or seven per cent of these parametrial exudates suppurate; the vast majority ultimately end in resolution. Cullen claims that this absorption can be hastened by incision just above Poupart's ligament, and drainage of the exudate between the folds of the broad ligament; our experience has shown us that incision and drainage of a solid exudate only prolong the reparative process. On the other hand the employment of heat for long periods by the use of the Gellhorn or Bier electric baker or the electric thermopad distinctly favors the absorption of an exudate by the production of a circulator stasis. After baring the abdomen and covering it with a bath towel, the baker is applied for one or two hours at a time, maintaining a heat of 140° F., or the thermopad may be held in place with a many tailed binder. Only when an abscess forms, as is shown by the evening elevations in temperature, by the increase in the polymorphonuclear count, and by fluctuation, is incision and drainage employed.

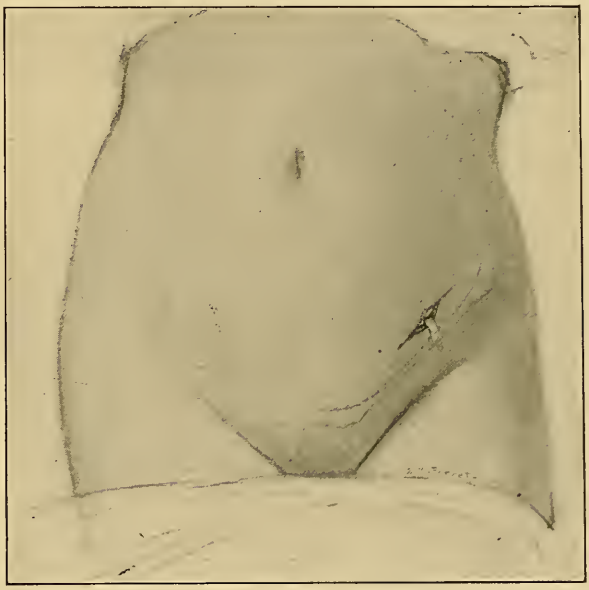

FIG. 35.—SITE OF INCISION JUST ABOVE POUPART'S LIGAMENT WITH A "CIGARETTE" DRAIN BETWEEN THE FOLDS OF THE BROAD LIGAMENT.

Before taking up the management of peritonitis, thrombophlebitis, and bacteriemia, a few words on the general supportive treatment of

the individual patient may not be amiss. Broadly speaking, *everything that can be done to improve the woman's general health will be an aid in combating the disease.* Fresh air, sunlight, an ample amount of easily digested food, and supportive stimulation, all contribute to the increase of her resisting powers. It is our habit to have our septic cases cared for on the roof, where they may remain day and night; the fresh air increases their appetite and favors sleep, while the sunlight increases the red cells and hemoglobin.

The fever seems to make less ravages in a patient who is treated in the open, than in one who has the same care in a ward. These patients

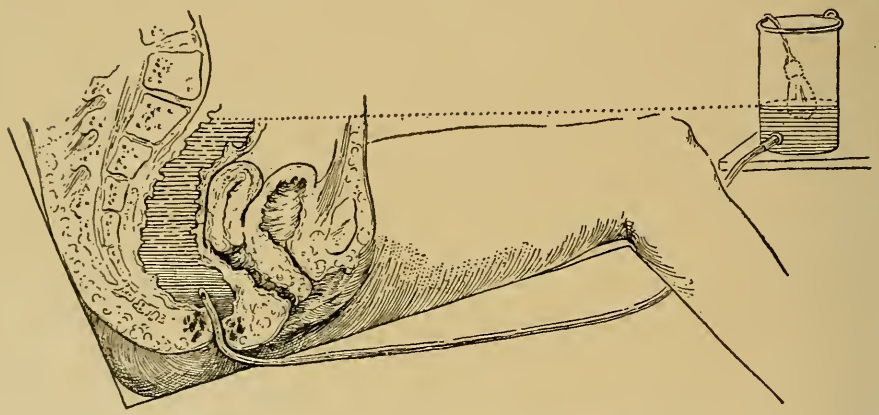

Fig. 36.—Harris Drip. Patient on Gatch Frame. Glucose-Soda Solution at Same Level in Both Can and Rectum.

sleep better, and sleep is a prime necessity in maintaining the nervous resistance. Sleeplessness, like pain, is depressing and fatiguing and sleep must be secured at all costs. We believe that a moderate amount of opium, either hypodermatically or by the rectum, to relieve pain, is less harmful than allowing the patient to suffer.

Little or nothing is done for the pyrexia, unless the temperature remains continuously high, when the fever is reduced by sponging or placing an ice cap on the head or over the heart, or an ice coil over the abdomen. While this does not actually reduce the temperature, it makes the patient feel more comfortable.

All catharsis is avoided in pelvic infections. In the acute stage, when it is necessary to empty the intestinal tract, the lower bowel is emptied daily by low enemata of small quantity; this causes less peristalsis than cathartics and is more agreeable to the patient. Diarrhea is one of nature's methods of carrying off the poison, thus ridding the system of toxins; however, repeated evacuations are exhausting and when con-

tinued, should be checked by saline irrigation, starch and opium enemata, and proper regulation of diet.

When the infection *spreads to the peritoneum* and there are *symptoms of peritoneal involvement, such as tympany and vomiting, all food should be withheld from the stomach,* and if the vomiting persists, the stomach should be lavaged. Nourishment may be supplied by enteroclysis, using a five per cent solution of glucose in a weak bicarbonate of soda solu-

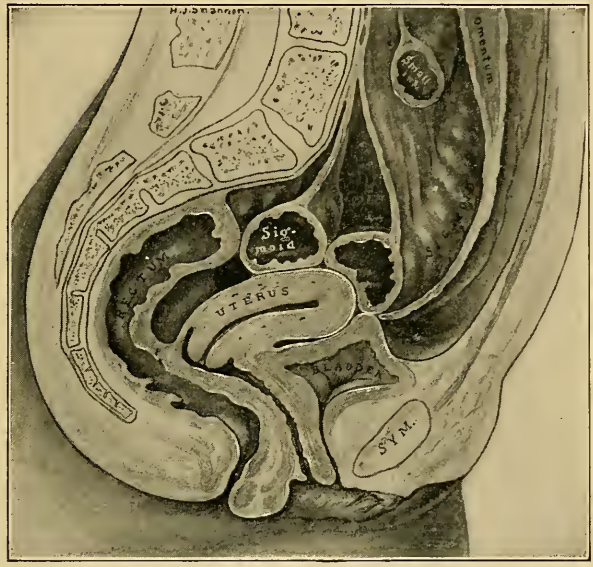

FIG. 37.—DIAGRAMMATIC SAGITTAL SECTION SHOWING ISOLATION OF PELVIS IN PELVIC INFLAMMATION. PATIENT IN FOWLER POSITION.

tion. If this is used by the Harris drip, a very considerable quantity of the solution will be taken up by the colon.

In perimetritis and pelvic peritonitis, we attempt to obtain localization by the elevated trunk posture of Fowler, the arrest of peristalsis with morphin, and the avoidance of cathartics. Morphin and an ice coil will generally control the pain unless the disease is progressive. The secretory functions are kept active by the continuous use of the Harris drip and daily hypodermoclysis of saline. If the peritonitis shows evidence of extension, as appears from the temperature remaining high, the pulse rate being accelerated, the leukocyte count being 30,000 or more, with a polymorphonuclear count of 90 per cent or more, we feel that a wide *cul de sac* incision and isolation of the pelvis with gauze, as suggested and practiced by the late Dr. Pryor, in conjunction with the Fowler elevated trunk posture, and the continuance of intestinal quiet

with morphin and ice, will frequently confine the infection to the pelvic peritoneum and prevent its extension to the general peritoneal cavity.

While the clinical picture of spreading peritonitis has been referred to before, little positive information can be obtained by vaginal examination.

Usually the abdomen is distended and the tenderness increased over a greater area, and the patient complains of increasing intestinal pain.

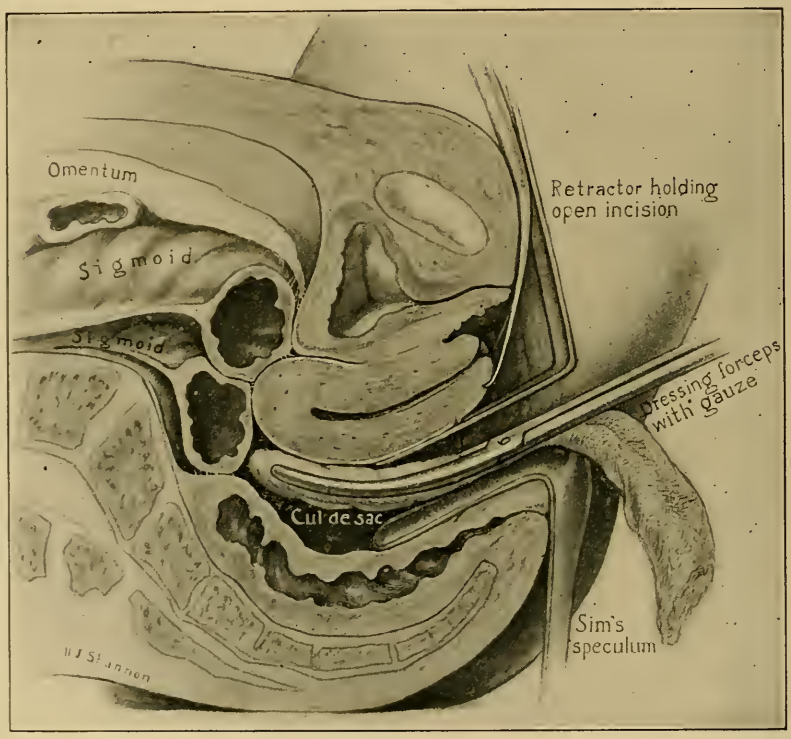

FIG. 38.—PLACING THE GAUZE ROLL DRAINS. THE POSTERIOR VAGINAL INCISION HELD OPEN WITH LONG BLADED RETRACTORS.

Vaginal examination seldom reveals more than local tenderness and exquisite sensitiveness on motion of the cervix; we have seldom found bulging of the *cul de sac,* notwithstanding this, on opening the *cul de sac,* we have invariably liberated a considerable quantity of seropurulent material, containing cocci. By widely opening the *cul de sac* and allowing this exudate to escape, and then isolating the pelvis by placing twisted gauze drains from pelvic wall to pelvic wall, we not only take care of the exudate already formed, but relieve the patient from the absorption of toxins. By the presence of the gauze we excite a local peritoneal reaction, which walls off the pelvis from the general abdominal

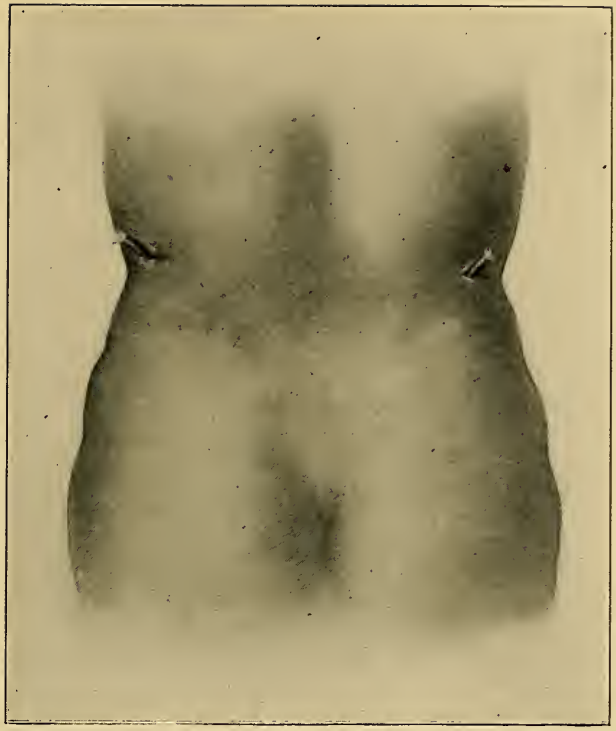

FIG. 39.—COUNTER INCISION AND DRAINAGE THROUGH THE LOINS IN PURULENT PERITONITIS.

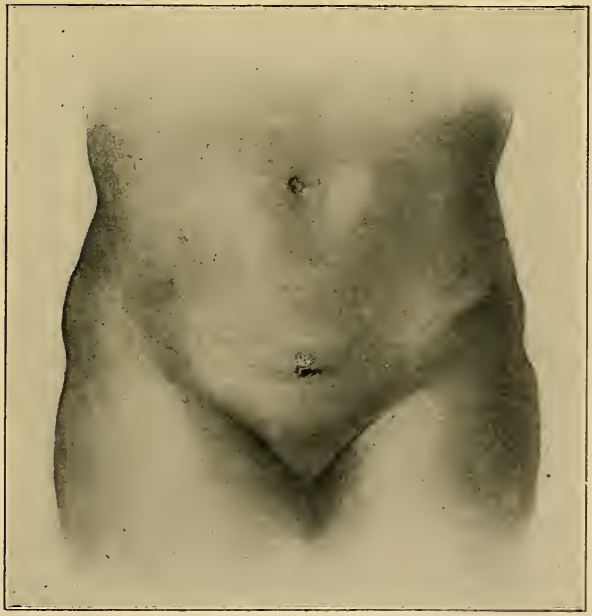

FIG. 40.—SUPRAPUBIC "STAB DRAIN" IN SPREADING PERITONITIS.

cavity, as the uterus is pushed forward and becomes adherent to the bladder and the loop of the sigmoid falls down and adheres to the fundus of the uterus and bladder. In this way the peritoneal reaction is confined to the visceral and parietal peritoneum within the pelvis. It must be understood that this radical method is not employed as routine, for the

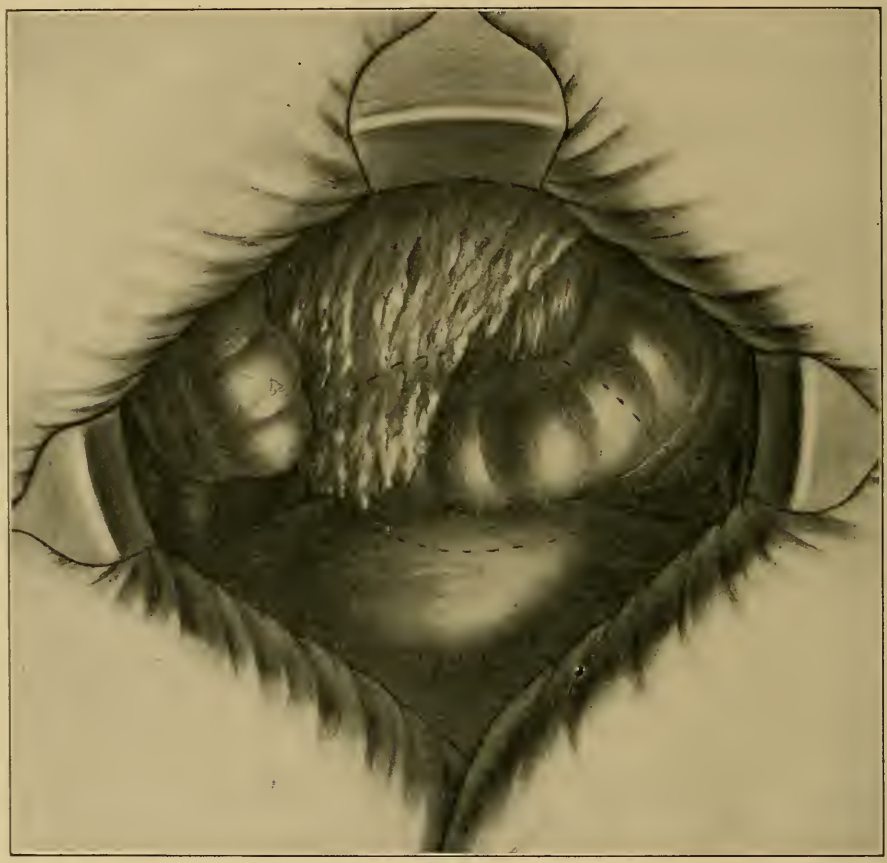

FIG. 41.—DRAWING FROM AUTOPSY AND OPERATING TABLE PATHOLOGY, SHOWING ISOLATION OF PELVIS IN CASES OF POSTABORTIVE PERITONITIS, FAVORED BY THE FOWLER ELEVATED TRUNK POSTURE.

majority of the cases of pelvic peritonitis and perimetritis tend to remain localized within the pelvis, and the exudate is absorbed.

If general peritonitis develops in the puerperium, there is no treatment which has given us much in a curative way. We regard the symptom complex of increased pulse rate, increased distention, abdominal pains and a polymorphonuclear count of 90 per cent or more as an indication for drainage. Early stab wound incision and drainage by the suprapubic route, with counter incisions and drains in the loins supple-

mented with posture, lavage, proctoclysis, and the arrest of peristalsis with morphin, have given practically no better result than the expectant plan. Altogether fifteen cases have been studied; seven were operated on; there were five deaths. Eight were treated by posture, lavage, proctoclysis, hypodermoclysis, morphin, and so forth, with six deaths. Many patients fail to localize the infective process in the pelvis, owing to the energetic measures employed by the attendant. *We have learned by bitter experience that puerperal and postabortal infective processes are actually extended by manual and instrumental manipulation.*

In bacteriemia, we have the presence of bacteria in the blood; these bacteria, with their toxins, produce a rapid dissolution in the cellular elements of the blood tissue, destroying the red cells as well as the leukocytes, and as they pass along the blood stream, through the heart, the kidneys, and the liver, produce degenerative changes in the heart muscle, cloudy swelling in the kidneys, and fatty degeneration in the liver; hence, it will be seen that, in order to combat bacteriemia, nature must offer a resistance so great as not to be overcome by the bacteria and their toxins.

Necessarily, the whole treatment hinges on our ability to increase the individual's resistance sufficiently to make the fight. Fresh air, sunlight, food, *repeated small blood transfusions,* and supportive stimulation, sum up the therapy.

Clinically, we must regard the blood as a tissue with all the characteristics of other tissues, hence it is obvious that the introduction of bacteria and their toxins into the blood stream must result either in a cell reaction or in a cell destruction. Bacteriologists have shown that blood serum makes a good culture medium for the culture of bacteria. If this is so in the laboratory, it is also true in the blood stream; hence there is no way by which nature can overcome the rapid propagation of bacteria, except by increasing the leukocytic resistance. The leukocyte is a bacterial enemy, and whether the patient recovers or dies depends upon whether the bacteria are killed by the leukocytes or the leukocytes destroyed by the bacteria.

Various attempts have been made by the profession to check bacterial growth, and to destroy the bacteria already in the blood by the introduction into the blood stream of antibactericides—various antiseptics, foreign material—by blood dilution and, finally, by the introduction of vegetable matter. Recently Potel has suggested injections of a 10 per cent solution of peptone in distilled water, using 10 c.cm. of the solution daily by intramuscular and intravenous injection. The latter, however, produces considerable reaction.

Chalfont and Miller report a series of streptococcic infections, which have yielded to the introduction of salvarsan into the blood. My own experience with salvarsan used exactly as recommended by Chalfont and Miller, in their paper, has not produced the results claimed by these writers; and the more one thinks of the actual pathological processes which take place in the blood stream after the introduction of hemolytic bacteria, the more one must wonder how the introduction of salvarsan can in any way affect the life and growth of these bacteria.

On the other hand, we all know that infection is arrested by the development of a leukocytosis. We know that the leukocyte is a bacterial enemy; we know that it is a struggle to determine the fittest, and whether the bacteria are killed by the leukocyte, or the leukocyte by the bacteria depends on the supply of each. It has, therefore, appealed to us that, notwithstanding the poor results (reported by Linderman and others), in the treatment of postoperative septicemias due to general surgical causes, that the use of small repeated blood transfusions offers the only plausible treatment in these blood infections.

Transfusion in infection would seem to serve the double purpose of lessening the secondary anemia and supplying normal active leukocytes for temporary defense, besides raising the blood pressure, which helps to restore functional activity of the organs bearing the brunt of the defense.

In a study of the poor results occurring among the surgical septicemias, we have become convinced that it is because the blood resistance is not *increased at a sufficiently early date, that transfusion, instead of being used in a rational way, has been used as a last resort.*

Many operators have been deluded into the belief that, when a large quantity of blood is poured into the blood stream, they get the effect from the quantity injected. Large transfusion not only often embarrasses the pulmonary circulation, but engorges the liver and spleen and thus interferes with their functional activity. We must appreciate that the effect of transfusion is but temporary, that the blood cells thrown in are rapidly destroyed, and that the real *effect is but a stimulation* of the tissues to increased cellular proliferation.

We have found that, within four or five days after a transfusion, the hemoglobin shows a marked reduction from the percentage obtained immediately after the operation, and that, while the leukocytes are immediately increased, they rapidly fall.

On this basis, accepting these clinical facts as truths, for they have been certified to by repeated observations, we have during the past year been studying the effects of repeated small transfusions of citrated blood

in bacterial invasions of the blood stream. So far, we have treated four cases of thrombophlebitis, with one death, and six cases of bacteriemia, with one death. The two fatal cases were, in our opinion, too far advanced, as the number of colonies of bacteria to the cubic centimeters were so numerous that it is doubtful if anything could have changed the outcome. In the fatal instance which occurred in thrombophlebitis, the hypogastric and ovarian vein had been tied before transfusions were given. This case, as have all of the others, showed a marked temporary improvement following the transfusion.

Small transfusions of citrated blood are given every third day, care being taken not to use the same donor for more than two transfusions. Quantities of 300-360 c.cm. are used, and given very slowly. The transfusion is preceded by a hypodermic of a third of a grain of morphin; we have found that this materially diminishes the severity of the reaction. The blood injections are best given in the morning, when the temperature is down. Our experience is limited, but, so far, has been such as to encourage us in the continuance of the method. Detailed blood studies have been made before and after each transfusion; the leukocyte count has been invariably increased and the blood pressure has been raised. Temporarily the red cells and hemoglobin have been increased; these, however, have shown rapid destruction in the succeeding forty-eight hours. *The pulse, in the favorable cases, has always improved in quality and rate,* whether the temperature has shown any favorable change or not.

The advantages of citrated blood are obvious; its simplicity, the fact that the patient may remain in bed, the absence of the donor, and naturally the lack of psychic disturbances.

Gas Bacillus Infection.—Symptomatology.—A brief word will suffice. We have all grades of infection from the mild toxemia, with only a slight rise of pulse and temperature, in the puerperium, to the rapidly fatal cases of gas sepsis. In the puerperal cases the symptomatology depends upon whether the uterine wall and the blood are invaded. If these gas builders once get beyond the confines of the genital tract and become mixed with streptococci, they usually evince severe invasive qualities. Leukocytosis is scant and the toxemia is rapid and prostrating. Vomiting and collapse are prominent symptoms, including hemolysis, jaundice, and bleeding into the vital organs and on the serous surfaces and skin. There are present rapid pulse and respiration and, toward the end, an ashy gray color to the skin, a progressive coldness of the body, beginning in the hands and feet, with often a clear intellect to the end. General body emphysema may be present.

Diagnosis.—This must be made bacteriologically. Any temperature in the puerperium, which seems to emanate from infection of the genital tract, should be checked up by intra-uterine cultures, grown both aërobically and anaërobically. It is important to make an early bacteriologic diagnosis, in order to start combative measures. The gas bacillus alone is harmless and ever present, but when combined with the streptococcus is rapidly virulent.

Prognosis depends upon the virulence of the infecting strain, its symbiosis with other organisms, the resistance of the patient, the amount of dead material in the uterus, the amount of trauma at labor. The fatal issue depends upon the invasion of the uterine wall.

Wright shows that measuring the antitryptic power and alkalinity of the blood aids in prognosis.

Treatment.—The treatment is local and general. Locally, what are we to do? Numerous authors have advised keeping the uterus open to the outside air by means of packing, drainage tubes, etc. Marvel saw five cases, all fatal except the last, in which he used intra-uterine irrigation, with peroxid of hydrogen.

Shall we remove the infected content of the uterus? In the abortion cases with bacillus aërogenes capsulatus in the discharge, good results are reported. If the dead material is the baby, as in my case, I would say, by all means, remove it. Large amounts of infected placental tissue should best be gently removed, though this may be dangerous in the presence of other infecting organisms, such as streptococcus, staphylococcus, etc. We have seen, however, that the germs grow in the dead tissue and increase their virulence, and large amounts of placenta tissue should be removed. Sir A. E. Wright suggests the early intravenous use of alkalis, and this would seem to be logical, though the measure has lost something in importance since we know that there is a true toxemia as well as an acidemia.

Our best hope for therapy lies in the use of the antitoxin serum elaborated by Bull. This serum should be given immediately upon finding the bacillus aërogenes capsulatus in the lochia. And our results will depend upon an early recognition of the condition. I cannot impress upon you too strongly the importance of the early bacteriological diagnosis, and the necessity in all cases for the immediate use of the serum.

CHAPTER VI

FIBROSIS UTERI, METRITIS, SUBINVOLUTION

Invasion of endometrium by infective bacteria—Gross pathology of chronic metritis, subinvolution, and uterine hypertrophy—Conditions which hinder normal involution—Some change in menstruation characteristic of metritis or subinvolution—Thrombokinase—Hemorrhage the most common symptom—Treatment by rest, hot douches, vaginal tamponade, galvanism, drugs, radium—Surgical treatment—Curettage—Hysterectomy.

Fibrosis uteri is a term which is applied to a pathological lesion directly attributable to infection of the uterus. It commonly occurs subsequent to repeated parturition or abortion, and therefore it is closely allied with *chronic metritis* and should be considered with it.

When the uterus is acutely infected, the endometrium becomes invaded by infective bacteria, such as the staphylococcus, streptococcus, colon, or mixed organisms, and there is a defensive inflammatory reaction on the part of the uterine tissues. The endometrium is a lymphoid structure without a submucosa, hence the prolongation of its tubular glands extends into the underlying muscular coat. Therefore, in the course of time, reaction to this invasion in the form of serum, round cells, plasma cells, and leukocytes is poured out into the stroma and the subjacent muscle structures.

The longer the infection is continued the greater is the number of round tissue cells which are deposited. This deposition of round tissue cells thickens the wall of the uterus and increases its gross bulk; and, by pressure, it destroys the muscle fibers, which are replaced by fibrous tissue. Therefore, when the process comes to the chronic state, we find, histologically, that the walls of the uterus are composed of an increase of yellow elastic, and fibrous tissue, as well as an increase in the gross bulk.

A further study of the pathological changes shows us that the endometrium is almost always in a state of glandular hypertrophy. The glands are larger, and dilated, and actually approach an early adenomatous growth. The arteries become thick walled and are surrounded by a large amount of elastic tissue, which inhibits the normal muscular contractions from controlling the blood vessels; consequently, bleeding is the most constant symptom.

Geist says that the same pathology may exist in women who have had repeated pregnancies, yet who have no definite history of infection; and claims that long continued subinvolution will explain the similarity. Our clinical experience is that *subinvolution is always a result of a mild degree of infection,* and therefore we must have some changes due to a long continued, low type inflammation; hence, the clinical substantiates the pathological findings.

The Gross Pathology of Chronic Metritis, Subinvolution, and Uterine Hypertrophy.—The gross specimen in these conditions, when examined macroscopically, shows the uterus to be symmetrically enlarged; its walls have a thickness of from three fourths to one and one fourth inches; the cavity has a depth of from four to five inches, and the whole organ is denser and harder than normal. On bisection, the wall looks "streaky" in the outer third. The "streakiness" is due to the muscle bundles being separated by large quantities of elastic and fibrous tissue. The vessels are enlarged and stand out on the cut surface; the endometrium is either very thin or excessively thick.

Dividing these uteri into the three types mentioned above, which we group clinically under the head of chronic metritis, Shaw has found in a microscopical study of 100 specimens, that 95 fell into the class of subinvolution, four were due to hypertrophy, and only one was the result of a true chronic inflammation.

To prove microscopically what occurs in chronic metritis, it is necessary to make use of a uterus which has never been pregnant, but in which there has been an acute or chronic inflammation, both uterine and perituterine.

In such a uterus, when the acute inflammation resolves, the inflammatory exudate is transformed into fibrous tissue, and the muscle tissue is also more or less transformed. So, in the uterus which has been the site of this inflammatory process, we should find a great increase in the amount of fibrous tissue, with an increase in the thickness and density of the uterine walls. The muscle bundles are diminished in size and separated by masses of fibrous tissue. In the earlier stages the tissues will be invaded with leukocytes and plasma cells. Later, these will have completely disappeared.

The effect of sepsis in the puerperium is apparently to delay involution, and it does not in itself produce any microscopic changes in the uterine wall, except those due to the persistence of a prolonged hyperemia.

For a clearer understanding of these processes, it will not be amiss to briefly review the normal process of involution.

Helme claims that there is no fatty degeneration in the muscle fibers, but simply a *diminution in the volume* of both the muscle fibers and the connective tissue. This change is brought about by a process which is probably chemical, a sort of peptonization of the tissues, which makes the contents of the muscle cells more soluble, so that they can pass with greater readiness into the lymph stream. This atrophy goes on simultaneously and equally in all parts of the organ. No group of degenerated cells is found among the healthy tissues. On the other hand, Goodall has found definite evidence of *fatty degeneration* in the muscle fibers.

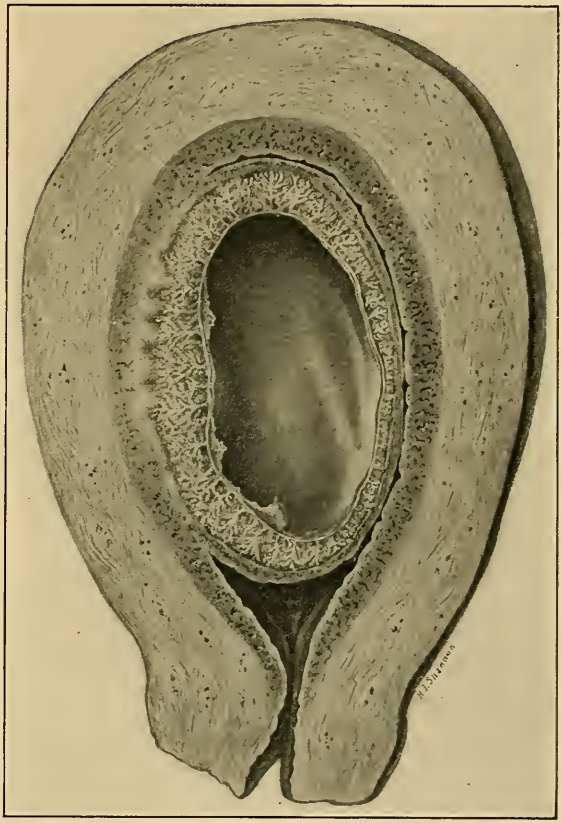

FIG. 42.—DRAWING FROM SPECIMEN IN AUTHOR'S COLLECTION SHOWING INCREASE IN CONSTITUENT ELEMENTS, THE THICKENED DECIDUA OF PREGNANCY AND EARLY PLACENTA FORMATION.

A review of Goodall's work deserves our consideration. He says "During pregnancy there is a great increase of all the constituent elements of the uterus. *The muscle fibers become enormously enlarged, while the fibrous and elastic tissue is also increased in amount;* but all through pregnancy *these three elements retain the same proportion* and distribution *as in the virgin uterus.* Coincidentally a great change takes place in the blood vessels of the organ. They enlarge sufficiently to supply the growing organ and convey the large amount of blood necessary for the sustenance of the fetus. All of the vessels enlarge, but especially those of the subplacental site, which become large sinuses.

"After labor the uterus undergoes a sudden tissue change. Soon after delivery, the fundus is at the umbilicus and the uterus weighs from one and one half to two and one half pounds, while in three weeks after

delivery the uterus weighs only from four to five ounces. This rapid change in size, weight and involution is completed in from eight to twelve weeks.

"Soon after delivery fatty degeneration occurs in the muscle fibers. This causes a great reduction in their size, while many are completely destroyed. Likewise, immediately after delivery there is a great reduction in the amount of blood conveyed by the uterine vessels, which reduction continues as the involution progresses.

"To meet this, definite changes occur in the vessel walls to reduce their lumina to their normal carrying capacity. *The vessels in the submucosal layer become blocked with thrombi.* In others, especially in the deeper layer of the uterus, a *ring of thrombus is formed within* the vessel which leaves its lumen still patent, but diminished in size. Coincident with this thrombosis, the fibrous tissue in the media of the vessel becomes markedly swollen and hyaline, and the elastica interna undergoes 'vitreous degeneration.' Instead of forming a thick black ring when stained with Weigert-Van Giesen solution, the elastica interna, which becomes enormously thickened, stains brick red in color and finally yellow.

"In this early stage we find some of the vessels filled with thrombi, others lined with thrombus, and others containing no thrombus at all. Most of the elastica interna is enormously swollen and has changed in color to brick red, bright red, or yellow. The media contains fibrous tissue, swollen in appearance, and hyaline, with free nuclei.

"The elastic fibrils in the media undergo 'vitreous degeneration,' then become swollen and change their staining properties in just the same way as did the elastica interna. The adventitia undergoes the same hyaline changes as the fibrous tissue of the media.

"The next step is for the hyaline tissue of the media to flow through the degenerated elastica interna and, where a thrombus is present, to convert it completely into hyaline tissue; when no thrombus is present this hyaline material flows around the sides of the vessel adjacent to the elastica interna, leaving only the *small lumen necessary.*

"In these vessels, which are filled with thrombus, the lumen becomes completely occluded with the hyaline material, which is gradually absorbed, so that the edges of the elastica interna gradually approximate and completely obliterate the vessel.

"In the vessels which contained only a layer of thrombus, or where the hyaline material flowed in without any previous clotting, a lumen is preserved, which now becomes lined by a layer of cells with large nuclei, and these form the endothelial lining of the new vessel. Coincidentally with this growth the hyaline material becomes invaded with

muscle and fibrous cells, which gradually convert the hyaline material into a muscle and fibrous ring, the media and adventitia of the new vessel. At this stage we find the new vessel surrounded by the old vessel wall, the old elastica interna being much swollen and in a stage of 'vitreous degeneration' and the old media containing swollen hyaline, fibrous, and elastic tissue.

"Between the old and new vessel walls is the interarterial space filled with hyaline material.

"When these new vessels are formed, the next important step in involution is the degeneration and absorption of the old vessel walls.

"Extensive fatty degeneration occurs in the old vessel walls which, if involution is complete, entirely destroys the muscle fibers of the old media, while at the same time the degenerated elastic and fibrous tissue of the old media and adventitia is absorbed. The old vessel is absorbed and the new vessel is left without any trace of the old surrounding it."

Conditions Which Hinder the Process of Normal Involution are:

1. Advancing age,
2. Frequent pregnancies,
3. General chronic and acute diseases,
4. Local pelvic disease,
5. Retained pieces of placenta and membranes,
6. Septic infection, often so mild that the patient apparently has a normal puerperium, except that involution is retarded.

When any of these conditions has operated to delay or stop the course of normal involution, degeneration of the muscular tissue of the uterine wall is arrested and the organ remains larger than when the normal process has gone on without interruption. The fibrous and elastic tissue throughout the wall has degenerated and become swollen, preparatory to being absorbed, but, the absorption process having been arrested, these swollen, fibrous and elastic tissues gradually regain their normal functions. Consequently, elastic and fibrous tissue is found in increased quantity between the muscle bundles, especially in the outer third of the uterus, and there is also a large amount of unabsorbed muscular tissue; these all contribute to increase the uterine bulk. New vessels have been formed and, according to the amount of diminution required for each vessel, several new arteries or one complete new artery will be found within the lumen of the old one.

Early in the puerperium these new vessels were surrounded by the old vessel wall, which underwent changes preparatory to absorption, the

elastica interna being enormously swollen, the fibrous and elastic fibers of the media swollen and hyaline. With the arrest of involution this old wall is not absorbed and the elastic and fibrous tissues regain their property, and so the *new vessels, or groups of new vessels, will now be found surrounded by thick slabs of elastic tissue and by the remains of the old media,* thickly impregnated with elastic tissue.

At first the uterine wall is soft, but later the tissues become more and more compact, making it feel firm and hard. In the walls of the veins much more elastic tissue will be found, especially in cases of subinvolution due to wasting diseases.

In some cases there have been alternating periods of involution and arrested involution. In these the uterine arteries will be found with alternating rings of muscular and elastic tissue. The endometrium is usually much thickened, to even eight or nine times the normal thickness. The glands are dilated and spiral in shape and may be increased in number, or the uterus may have very little endometrium.. To summarize the changes due to subinvolution:

The *uterus* is *symmetrically enlarged and hard; on cut surface* the *vessels,* with their *surrounding tissues,* are *strikingly visible* and *stand out* from the *surface;* the *endometrium* is *thickened* or *diminished;* the microscope shows some increase in the amount of *fibrous* and *elastic tissue* between the *muscle fibers,* with a large accumulation of elastic tissue around the arteries and groups of arteries.

Some change in menstruation is strikingly characteristic of metritis or subinvolution; hence, it may be well to study the origin of the hemorrhage in these conditions. This bleeding is in all probability due to the collection of fibrous and elastic tissue around the arteries, which prevents the muscular tissue of the uterus from exercising its normal control over these vessels, and thus from regulating the amount of blood which passes through them.

It has already been mentioned that the endometrium may be hypertrophic or atrophic. In the hypertrophic variety, the hemorrhage may be due to the changes in the endometrium, though to what extent they PRODUCE OR INCREASE the hemorrhage in these cases we cannot at present be certain.

The studies of Beckwith and Whitehouse seem to throw new light on this subject. When blood is poured out into a tissue, as into the endometrium during menstruation, it comes into contact with a substance called "thrombokinase." This substance, which varies in amount in different tissues, promotes the union of thrombogen and the calcium salts to produce thrombin; the latter then exercises a quantitative action

upon the fibrinogen, converting it into fibrin, the proteid which forms the organic base of all clot.

"Thrombokinase" is especially plentiful in the endometrium and is nature's provision for checking excessive hemorrhage in an organ which is subject to periodic hemorrhages. The clot formed by the action of this "thrombokinase" blocks the vessels and in a healthy endometrium prevents excessive hemorrhages. If from any cause the "thrombokinase" is diminished in amount, clotting of the blood will be delayed and there will be severe hemorrhage.

Whitehouse considers this to be the cause of excessive hemorrhage in uteri which have an atrophic endometrium, as this atrophied structure does not produce the necessary amount of "thrombokinase."

Besides "thrombokinase," there is another substance produced in the endometrium, "thrombolysin," which has a marked effect on the production of hemorrhage.

Owing to the "thrombokinase," clots are found in the uterus, but in the menstrual blood found in the vagina in women with normal menstruation there are no clots; this is due to "thrombolysin," which has the power of dissolving blood clot, and in the normal uterus dissolves the uterine clot formed by the "thrombokinase" before it reaches the vagina.

In a normal uterus the amounts of "thrombokinase" and "thrombolysin" are balanced, the clotting is produced in the uterus by the "thrombokinase" and excessive hemorrhage checked, while later these same clots are dissolved by the "thrombolysin" and the dark fluid blood passes through the os into the vagina.

If the amount of "thrombolysin" is in excess, the blood clot will be too rapidly dissolved and the blood vessels again opened up with a resulting hemorrhage.

From these experiments of Whitehouse it would seem that hemorrhages in metritis may be due either to a diminution in the amount of "thrombokinase" or an excessive amount of "thrombolysin," the supply of the ferments depending apparently on the cellular activity of the endometrium.

In subinvolution the hemorrhage may be explained, in addition to these chemical phenomena, by the want of muscular control over the vessels, due to the excess of elastic tissue about them.

Etiology.—Fibrosis is commonly found in multiparae between the ages of thirty-five and forty-five, almost never in the virgin, unless there has been a history of previous chronic inflammation, and there is commonly some relaxation of the pelvic floor.

Bennett states that "to this class belong a large proportion of the pop-

ulation of the sofa and bath chair type, nervous, debilitated, and dyspeptic females, who wander from medical man to medical man seeking relief from weakness."

Although this condition is more frequent toward the latter end of sexual activity, it is at times found in early life. Watson reports a patient of twenty-three who required hysterectomy.

Symptoms and Signs.—There are three cardinal symptoms, (1) hemorrhage, (2) leukorrhea, and (3) pain; most patients complain of a combination of these.

The uterus is symmetrically enlarged and hard and is usually insensitive to manipulation. Section will show that there are no fibromyomatous nodules. The weight tends to cause the uterus to antevert, retrovert, or prolapse. In about 25 per cent the uteri are retroverted and prolapsed. Some degree of chronic endocervicitis is always associated. This involvement of the cervix tends, through its lymphatic drainage by way of the uterosacral, to produce a mild uterosacral cellulitis.

The heavy uterus drags on the tender uterosacrals, and backache, constant and tiring, is the result.

The most common symptom however is hemorrhage. The hemorrhage may take the form of metrorrhagia, in which case the patient has a more or less constant, bloody discharge from the uterus, which increases in amount at the time of the regular periods. The bleeding may appear as a menorrhagia, the flow being more profuse and prolonged at each menstrual date, until sometimes the intermenstrual interval is reduced to four or six days.

Secondary anemia follows; this produces the train of general symptoms complained of by these chronic invalids, for a poor state of health explains the tiredness, indigestion, flatulence, headache, etc., to which they are subject.

Pain, as a symptom, may occur in two types; first, as pain occurring at the menstrual period, i.e., dysmenorrhea, bearing down and cramp-like in character; second, as a chronic aching pain in the back, loins, and iliac regions. Usually the symptoms of lumbosacral backache date from a confinement or an abortion. Dysmenorrhea is commonly the result of a febrile confinement or abortion.

In that rare form of chronic metritis in the nulliparae due to tissue hypertrophy, the dysmenorrhea may date from puberty and is evidenced by the occurrence of excessive uterine contractions. Here the chronic pain in the back and iliac regions is constant. This is worse on standing and exertion, due to the drag of the heavy uterus on its uterosacral ligaments. The leukorrhea is not a constant symptom, nor is it ordi-

narily as distressing as in the condition of infective metritis, except that it may be thin, acrid and irritating and produce an intractable pruritus.

The differential diagnosis of metritis must be made from early pregnancy, small fibromyomata, carcinoma of the uterine body, and uterine adenomyoma.

A patient with chronic metritis who has recently become pregnant will often give a history of menorrhagia with no period of amenorrhea; but on close questioning, there is always something atypical.

A two months pregnant uterus is about the size of a chronic metritic uterus, but it intermittently contracts, and is always *at some time of softer consistence. Its shape changes on contraction and during relaxation.* There is some softening of the cervix, an increased and changed secretion from the cervix, a dusky hue about the cervix and vaginal vault, and increased pulsation in the fornices. An Abderhalden test may aid in the diagnosis.

A small interstitial fibroid may present extreme difficulties in differential diagnosis. Irregularity in contour points to fibromyoma, a sound may show tortuosity of the canal, and will give an accurate appreciation of the position of the fundus. Examination under anesthesia, in stout women, will aid in the diagnosis. *Hysterotomy may be necessary to definitely determine whether we are dealing with a metritis or a tumor.*

The differentiation between metritis and carcinoma of the body is the most important diagnosis the gynecologist has to make. *No question of such moment can be decided except with the aid of the curet and a microscopic examination of the uterine scrapings, by a competent* pathologist.

Prognosis.—The prognosis of fibrosis, metritis, and subinvolution of the uterus is not serious as to life but tends to invalid the woman, owing to the large and continuous blood loss which she is forced to sustain. These conditions tend to prolong or postpone the menopause. Secondary anemia, which necessarily follows, diminishes her natural resistance and makes her a poor operative risk, producing, as it does, changes in the heart muscles and kidney functions.

Treatment.—The indications for treatment are:

(1) the control of hemorrhage, and
(2) reduction in the size of the uterus.

Medical Treatment Consists of:

(a) rest,
(b) hot douches,

(c) vaginal tamponade
(d) galvanism, and the
(e) administration of certain drugs.

(*a*) *Rest.*—The patient should remain in bed during menstruation and have the bowels emptied with enemata and avoid excitement or exercise. Observance of these rules usually diminishes the amount of blood lost and necessarily conserves the woman's strength.

(*b*) *Hot Douches.*—One or two gallons of hot water, at a temperature of 120° F., may be used twice a day. The douche is best given in the recumbent position, with the Davidson syringe, to insure the depletion of the pelvis; this changes the circulation in the uterus and stimulates its contractions.

(*c*) *Vaginal Tamponade or Pack.*—When properly applied this will temporarily control the blood loss, *and is the emergency method.* The patient should be in the Sims or knee-chest position, and the perineum retracted with a Sims speculum; then, after cleaning the vagina with one-half per cent lysol solution, the vagina is firmly packed with two inch wide packing gauze. The gauze should be placed in the fornices, against the cervix, and then made to fill the entire vagina. Moist gauze controls hemorrhage better than dry gauze. For when it is placed in position, it maintains its bulk and therefore maintains an even pressure.

(*d*) *Galvanism.*—It is claimed that, when the positive pole of the galvanic current is introduced into the cavity of the uterus and the negative pole is placed over the sacrum or pelvis, the current will stimulate the uterus to contraction and thus relieve the circulatory stasis, empty the uterine veins, and so diminish the amount of the menstrual flow. Our experience with galvanism for the control of hemorrhage in fibrosis has not encouraged us to be enthusiastic as to its curative value.

(*e*) *Drugs.*—Of the drugs used to check hemorrhage in this condition, thyroid extract, pituitary extract, ergot (or its derivatives), stypticin, styptol, etc., are the ones in common use. All have some value in controlling the bleeding, but result in no permanent cure, for the pathology shows that the minute changes in the muscle bundles and about the blood vessels are such that internal medication cannot produce any permanent good. Only by removal of the organs, or obstruction of its blood supply, can we hope to permanently stop the hemorrhage.

Surgical Treatment.—Under this head we have to consider:

(1) curettage,
(2) radium,
(3) hysterectomy.

(1) Every woman of thirty-five or forty who presents herself with anomalous uterine bleeding, after excluding the possibility of a condition of the pregnant state, is entitled to a diagnostic curettage; for, only by a microscopic examination of the removed endometrium can a positive diagnosis be made of the benignancy or malignancy of the uterine enlargement.

Curettage of the uterus will temporarily check the hemorrhage of fibrosis or subinvolution; for some part of the bleeding comes from the hypertrophied endometrium so frequently present in the early stages of this condition. But, as the actual cause of the hemorrhage is not in the thickened endometrium, or the atrophy of the glands, with changes in the gland products, but in the histologic changes in the uterine walls and in and about the blood vessels, little curative value can be expected from removing the endometrium with the curet. After curettage the woman may not bleed for one or two periods, and then the hemorrhage will return as a menorrhagia or metrorrhagia, or both, often with increased severity.

(2) *Radium will control the hemorrhage from fibrosis, metritis, and subinvolution in one hundred per cent. of selected cases* and reduce the size of the uterus to that of the senile organ; unfortunately, not all cases of fibrosis, metritis, and subinvolution are amenable to cure by radium, and its use is *contra-indicated in pelves which have previously been the seat of extensive inflammatory processes,* pelvic peritonitis, chronic salpingitis, etc. However, if the large, smooth, hard, uterus is freely movable and shows no para- or perimetrial or adnexal complications, radium may be safely used, and used with a knowledge that by an irradiation of twenty to twenty-four hours the hemorrhage will be checked, the leukorrhea will cease, and the uterus will atrophy.

No anesthetic is necessary, but the patient is given a third of a grain of morphia hypodermatically one hour before introducing the radium; and, after the vulva, vagina, and cervical portio have been properly prepared, the woman is placed in the lithotomy position and the cervix carefully dilated with Hegar's sounds to a size sufficient to allow the introduction of the radium capsule. Fifty to seventy-five milligrams of the radium salt, incased in a capsule of glass, an envelope of silver, and a brass filter, to remove the irritating β rays of the radium, are now introduced into the uterine cavity and retained in the uterus by plac-

ing a narrow gauze pack in the cervix, which in turn is supported by a firm gauze vagina pack. This technic prevents the expulsion from the uterus of the radium capsule before the time for its withdrawal, and thus prevents burning of the vagina, as well as the danger of its expulsion and loss.

The radium should be removed in twenty to twenty-four hours, as 1,000-1,500 milligram hours are sufficient to check most uterine hemorrhages occurring from this cause; following the removal of the radium, there is a serosanguinous discharge from the cavity of the uterus. This continues for five or six days, when the character changes to a seropurulent leukorrhea, which entirely ceases in the course of three weeks. In two months time the size of the uterus will have diminished to one-half the size it was prior to the irradiation, and in six months it will be senile, unless there are intramural or subperitoneal fibromata in the walls.

Kelly, Clark, and Miller all endorse this treatment of hemorrhages due to subinvolution or fibrosis by the application of radium to the interior of the uterine body.

(3) In the light of our present day pathology, the field for hysterectomy, in the cure of subinvolution, metritis, and fibrosis, is limited to those cases where there exists extensive actual trauma of the cervix or lower uterine segment, and those cases where the woman has been the subject of subacute or chronic pelvic inflammation, with recurring exacerbations. Such cases not only drain the woman's vitality by repeated bleeding, but render her an invalid with pelvic distress.

Total hysterectomy should be done, in order that all foci of infection in the cervix may be removed, but as these patients usually have a higher blood pressure than other women of their age, we feel it *is important to conserve the ovarian function,* for total removal of the sex glands breaks the harmonious and interdependent relation of the internal secretions and subjects the woman to increased nervous phenomena, *brought* about by *the uncurbed action of the thyroid, adrenal, and pituitary glands.*

CHAPTER VII

SALPINGITIS

Four routes of infection—Sepsis and gonorrhea the most common causes of salpingitis—Acute salpingitis—Hydrosalpinx—Clinical phenomena—Significance of the history—Differential diagnosis—Physical signs of pyosalpinx—Hydrosalpinx—Hematosalpinx—Gonorrheal salpingitis—Treatment of acute salpingitis—Technic of vaginal section (posterior colpotomy)—Treatment of chronic salpingitis—Salpingostomy—Technic of salpingectomy—Radical treatment—Technic.

Salpingitis is an inflammation of the fallopian tube, which is nearly always secondary to infection of the uterus or of the peritoneum.

The infecting organisms may reach the tube by four different routes.

1. They may gain access to the lumen of the tube from the interior of the uterus, as in acute gonorrheal infection of the endometrium.

2. They may reach the tube from the peritoneal cavity by way of the abdominal ostium, as in streptococcic and staphylococcic cellulitis and peritonitis, following childbirth and abortion. By this route the bacteria may be sucked in from the peritoneum by the ciliary current at the ostium, in which case the tubal infection is secondary to the peritonitis, or they may produce an endosalpingitis by extension through the lymph channels of the broad ligament.

3. They may gain access through the tube wall, when intestinal adhesions are present, as in the peritonitis following appendicular and intestinal perforations.

4. And finally, bacteria may invade the tube through the blood stream or lymph channels, as is the case in primary tuberculous salpingitis.

Sepsis and gonorrhea are the most common causes of tubal inflammation, and various microörganisms may be found. *The gonococcus is the organism most frequently met with, producing from forty to fifty per cent of all tubal infections.* The infection in these cases always ascends from the interior of the uterus. Infection by the streptococcus and staphylococcus is also generally an invasion from below. These bacteria excite a marked inflammatory reaction, causing a seroplastic exudate, which is poured out by the perisalpinx and peritoneum, with

formation of adhesions to the adjacent bowel. This in turn permits a secondary infection by the colon bacillus.

Salpingitis occurring before puberty is always gonorrheal or tuberculous in origin, although it is possible for a child to have salpingitis following the exanthemata. The inflammation is usually bilateral, although one tube may be involved at a time, and each may have a different pathology. Owing to the anatomical relations of the tube, it is seldom that a salpingitis remains localized. It becomes a part of an inflammation of the pelvic tissues, the infection extending to the adjacent ovaries, peritoneum, and contiguous pelvic structures. Not until the infection becomes subacute or chronic can the tubal pathology be definitely identified.

Acute Salpingitis.—It has already been stated that the infecting organism may be the gonococcus, the streptococcus, the pneumococcus, or the colon bacillus, either alone or in combination. *Mixed infections* have the most serious effect and produce the greatest tissue reaction in the tubal structures, and *invariably leave some permanent pathology.*

Pathology.—The invading organism first excites an inflammatory reaction in the tubal mucous membrane, but finally all the coats of the tube become involved. The mucosa becomes swollen and edematous, and there is a hypersecretion of mucus; serum is poured out into the underlying tissues, the surface epithelium proliferates, and, if the inflammation continues, dies and desquamates; as a consequence, in the ampulla and infundibulum, the folds become so thickened that they impinge upon each other, and the plicae, devoid of their epithelium, adhere to one another and practically block the lumen. Coincidentally, the muscular and peritoneal coats of the tube become edematous and infiltrated with inflammatory cells, and the fimbriated extremity is swollen; so that, at this time, histologic examination of the tube will show not only an endosalpingitis, but a myosalpingitis and perisalpingitis.

The edema and infection of the peritoneal coat causes swelling of the endothelial cells, and a serous or plastic exudate is poured out on the serosal surface, which leads to the *formation of visceral adhesions.*

The swelling of the mucosa of the fimbria may cause an inversion of the fimbria and adhesion of their peritoneal surfaces, thus closing the abdominal ostium; or, there may be pouting of the mucosa, which will prevent complete closure of the abdominal end, and thus allow the infection to spread to the adjacent peritoneum and ovary; or, an exudation, with adhesions between the tube and ovary, and between the tube and adjacent bowel, or with the pelvic peritoneum resulting. *Adhesion always more or less completely closes the lumen and temporarily checks*

the infective process. Closure of the abdominal ostium by adhesion of the peritoneal surfaces of the fimbria, or by adhesion to adjacent structures, always distorts and displaces the tube.

The most common displacement of the tube is downward, where it becomes adherent to the posterior surface of the broad ligament, or attached, in the *cul de sac* of Douglas, to the uterosacral ligament. As it drops downward and backward and inward, *it envelops the ovary,* burying it between the posterior surface of the broad ligament and the mesosalpinx. The nearby pelvic structures are often involved in the

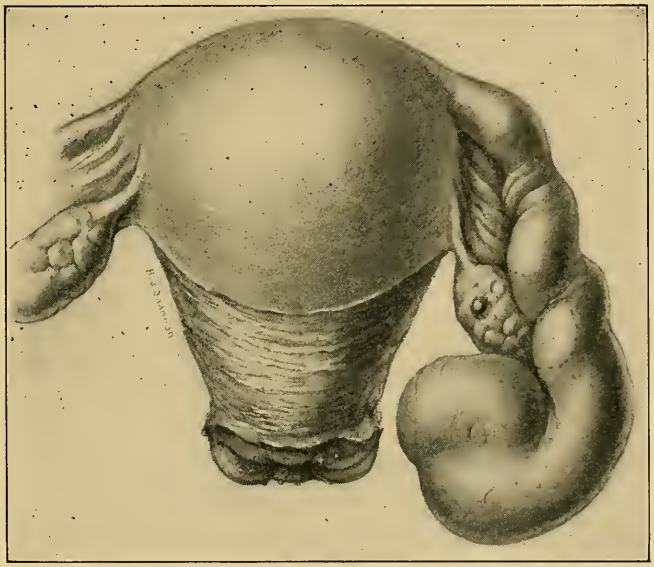

Fig. 43.—A Pyosalpinx Showing How the Tube Rolls Over the Ovary and Drops into the Cul de Sac.

infection, especially the ovary, and secondary collections of pus may form in the peritoneum. *Most pelvic inflammations originate in purulent salpingitis.*

To naked eye inspection, the tube is thicker, more tortuous, and of a brighter red color than normal. If the fimbriated end is free, pus may be seen escaping from the abdominal ostium.

The termination of acute salpingitis may be in resolution, as the inflammation may subside completely, leaving behind only a slight fibrous thickening of the tube wall and a few adhesions about the fimbriated extremity.

In our experience, gonorrheal infections, uncomplicated by other cocci, frequently terminate in complete regeneration of the tube. Several of our cases have subsequently become pregnant and have been delivered

of healthy children. The tubal inflammations which complicate postpartal or postabortal infections, particularly those in which cellular or peritoneal inflammations have been present, are not apt to regenerate so completely. *Mixed infections take longer to subside and always permanently damage the tube.* Pregnancy is unlikely to follow in this group. Sterility is the rule.

In other cases, as the result of the desquamation of the surface epithelium, adhesions may form between the plicae uniting the folds of mucous membrane; this narrows the lumen of the tube, or may even cause a complete obstruction, so that the tube may be divided into several compartments, each containing pus.

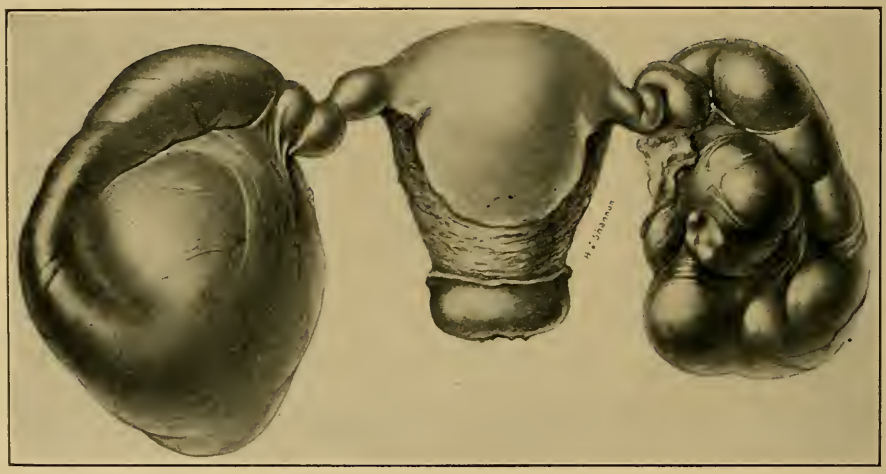

Fig. 44.—Double Hydrosalpinx from the Author's Collection.

The acute inflammation may pass into a subacute or chronic inflammation, and, as a result, the walls of the tube become greatly thickened; therefore, it may be increased in thickness to the size of the thumb and become tortuous, or the lumen may be dilated and filled with mucoid or purulent contents. In other words, a sactosalpinx is formed which may contain serum and mucus, blood or pus. These tubal swellings result from closure of the abdominal ostium by adhesion of the peritoneal surface of the fimbriae, or by adhesive attachment of the fimbriae to the peritoneum or ovary; in either event, to form a sactosalpinx, the abdominal end must be closed. Such closure may in time result in a hydrosalpinx or pyosalpinx.

A hydrosalpinx is a thin walled, translucent, retort shaped tumor containing a serous accumulation. The outer end of the tube is always closed; the uterine end may or may not be patent.

The narrow isthmic portion next to the uterus is practically undilated and remains of comparatively normal thickness. There is, however, always a cell proliferation into the muscle about the isthmic or interstitial portions, forming the so-called "isthmic node." The ampulla and infundibulum more readily dilate, hence the (retort shaped) form of these tubal sacs. Tubes distended in this way may attain a very large size, forming tumors the size of a grape fruit or larger, or they may intermittently empty themselves through the uterus.

As a result of intestinal adhesions to the tube, there is often a secondary infection by the bacillus coli; when this occurs, the original infecting organism frequently dies out, and after a time the bacillus coli also

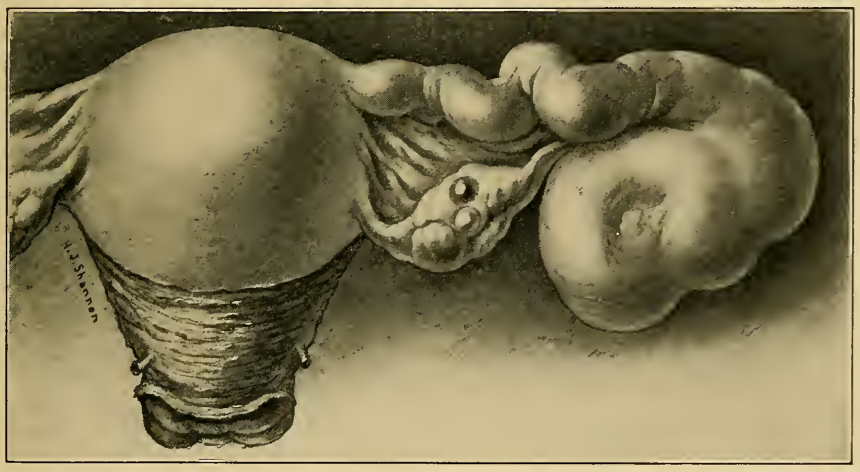

FIG. 45.—SPECIMEN OF PYOSALPINX FROM THE AUTHOR'S COLLECTION SHOWING INFLAMMATORY NODES IN THE ISTHMUS.

dies, and there is left a tube distended with pus, often foul smelling, but sterile. These purulent accumulations within the tube are called pyosalpinx. The fact that the pus in these cases may become sterile, is of great clinical importance, as, not infrequently, during the removal of such a tumor, more or less of its content is spilled over the peritoneal surface; but, as the *contained pus is sterile in from sixty to seventy per cent of the cases,* the soiling gives rise to little or no trouble.

Hyde's work has shown that *in gonorrheal infections the contained pus is sterile in from six weeks to three months, but the streptococcus may live in the tissues for years.* Operation on these tubes of streptococcic origin is always serious, as the liberation of the cocci may excite a fatal streptococcic peritonitis, while gonorrheal infection tends to remain localized within the pelvic basin.

The tube may become adherent and rupture into the peritoneal

cavity or into the adjacent viscera. Rupture into the peritoneal cavity is *always attended with the symptoms and physical signs of an abdominal calamity,* i.e., severe abdominal pain, shock and evidence of peritoneal reaction, as shown by tension, tenderness, tympany and a high leukocyte count, together with a high polymorphonuclear percentage; or, the escaping pus may, as the result of adhesions, become encysted in the true pelvis and form a pelvic abscess.

Clinical Phenomena.—The mode of onset varies with the nature and virulence of the infecting organism. In some cases it is very sudden, while in others the onset is more gradual. There is *acute pain in the lower abdomen,* sometimes in the midline, but more often in the

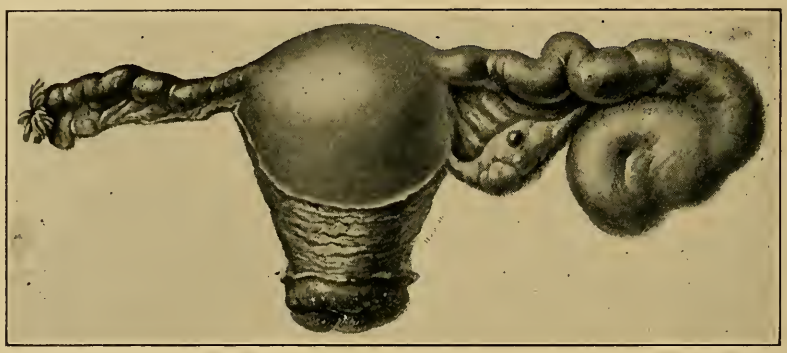

Fig. 46.—Suppurative Salpingitis on One Side with a Pyosalpinx on the Other.

lower quadrant over the side affected. *The pulse is increased in rate* and there is an *elevation of the temperature.* Owing to the muscular spasm over the affected tube, the lower abdomen does not move with respiration as freely as normal and, as a consequence, there are "still areas" over this region and tension and tenderness on palpation. *In acute cases it is difficult to dissociate the symptoms of salpingitis from those of the acute endometritis and peritonitis accompanying the tubal infection.*

The Significance of History.—The antecedent history of urethritis or of recent marriage and the occurrence of leukorrhea is as suggestive of gonorrhea, as the history of labor or abortion is of sepsis.

Symptoms.—Often the systemic disturbance is marked at the onset. There is sudden pelvic and abdominal pain and a rise of pulse and temperature proportionate to the virulence of the local process. Frequently the symptoms of general acute pelvic inflammation make an anatomical diagnosis impossible; but as the acute process subsides, a more exact "geographical" location of the primary focus can be made. There is always a *mucopurulent leukorrhea,* for the circulatory stasis in the uterus and cervix persists until all of the contiguous exudate has disappeared.

Furthermore, the cervix remains swollen and sensitive for a long period and, in consequence its *infected glands pour out an excessive muco-purulent discharge.* This leukorrhea is accompanied by pain and tenderness in the region of the affected tube, and this is more marked when the exudate in the peritoneum includes the ovary. The *pain is often intermittent or colicky* and depends somewhat on the state of the intestinal tract. *Intestinal activity always increases the pain.* There is *lumbosacral backache* because of the involvement of the peritoneum covering the uterosacral ligaments.

There may be *dysuria* and *dyschezia,* the latter being dependent upon the extent of the peritoneal exudate and the position of the tubal tumor. As the uterosacrals are always involved in tubal inflammations, one can readily see how, as the fecal mass passes between these swollen cellular bands, defecation may become exceedingly painful.

Menstruation is disturbed, owing to the increased congestion coincident with the parametrial and perimetrial inflammation. The bleeding may be too frequent or too profuse, and is *always attended with premenstrual and comenstrual pain.* There is usually *over the lower abdomen a premenstrual soreness,* which is due to the chronic peritoneal irritation. If the tumor mass is large or the peritoneal exudate extensive, vesical and rectal tenesmus is added to the symptom complex.

Intestinal tympany is more or less constant in all acute and subacute tubal inflammations, owing to the frequency of ileal, colonic, and sigmoidal adhesions, which produce paretic areas and interfere with normal peristalsis.

In chronic cases leukorrhea, premenstrual pain, dyspareunia, and sterility, alone or in combination, are the symptoms for which the patient seeks relief. All of the symptoms are aggravated by walking and standing.

Recurrent attacks of pelvic peritonitis are *characteristic of subacute tubal inflammation* and occur most frequently at or near the menstrual periods. These exacerbations are comparatively common in the early stages of chronic salpingitis, as in many cases the abdominal ostium is not entirely closed and leakage occurs, thus setting up renewed reactions in the adjacent peritoneum. Ultimately, however, the abdominal ostium becomes sealed by inversion of the fimbriae or by adhesions to the contiguous ovary or peritoneum, and then the tube becomes quiescent. With these local symptoms there is general debility, loss of weight, digestive and nervous disturbances. Continued local pain and infection not only cause general discomfort, but are bound to undermine the general health of the woman.

Leukorrhea, menstrual disturbances, dysmenorrhea, sterility, and intermenstrual pain are the principal symptoms. The pelvic pain usually subsides when the menstrual flow has become established.

The symptoms are not necessarily marked in chronic infection of the tubes, for *time effects a symptomatic cure in a large number of cases.*

Physical Signs.—On abdominal examination, in the acute stage there will be some distention with tension and tenderness on pressure over the lower quadrants. After the acute stage has passed, we may find nothing except moderate intestinal distension and tenderness on pressure over Morris's points. Tenderness over both Morris's points is always significant of pelvic inflammation, for the lymphatic drainage from the tubes is directed into the lumbar glands, along the vertebral column, over which these points are located.

On abdominovaginal or abdominorectal examination, the tube or tubal mass may usually be found as a thickened tender tumor at the side of and frequently behind, rarely in front of, the uterus. It is generally fixed. The uterus is larger than normal and more or less fixed by the associated *pelvic peritonitis and uterosacral parametritis,* which are *usually constant in all tubal inflammations.* This explains the pain caused by moving the uterus. The tube and ovary and often all of the other pelvic contents are so matted together into one conglomerate mass, that an anatomic diagnosis from the physical findings is absolutely impossible.

In tuberculous salpingitis there may be evidence of tuberculosis in other organs, or the tubercle bacilli may be found in the uterine discharge. Usually however the diagnosis cannot be made until the tube is removed and its contents examined and microscopic study made of sections taken from the diseased areas.

Differential Diagnosis.—It is sometimes difficult to differentiate right sided tubal inflammation from appendicitis, for abdominal operation has shown that appendectomy has been performed in ten to fifteen per cent of cases where tubal disease is the existing pathology. This is readily understood when we remember that the appendix may become adherent in the pelvis, and thus become a complicating factor in pelvic inflammation.

In *appendicitis,* both the *history and physical signs are so definite* that, if proper credence be given to them and their sequence is duly considered, it would appear that a *differential diagnosis should be possible* in almost every case.

J. B. Murphy, in a study of two thousand cases of acute appendicitis, found that the symptom sequence was always as follows: acute abdominal pain, most frequently referred to the region of the umbilicus or

epigastrium, followed by vomiting, the vomitus consisting of the contents of the stomach; this in turn is followed by local pain in the region of the appendix and a slight rise in temperature and pulse rate.

Physical examination at this time will show tension and tenderness in the right lower quadrant, with accentuated tenderness over both McBurney's and Morris's points (right). The blood count always shows a leukocytosis with a relatively high polymorphonuclear percentage.

When this sequence did not occur, Murphy found that the abdominal condition was due to some other lesion than an inflamed appendix. Further diagnostic aid may be had by making *downward pressure* with the hand *over the colon,* gently forcing the cecal contents toward the appendix. This *always causes pain in appendicitis.* Tenderness over the right Morris point, which is located one inch and a half below and one inch to the right of the umbilicus, over the lumbar glands, is also suggestive in appendicitis. While sensitiveness at Hänk's point, which is situated an inch and a half to the right and an inch above the umbilicus, being also over the lumbar glands, is found to be present in most cases of appendicitis.

Muscular tension is generally more marked in the presence of appendiceal inflammation than in an inflammation of pelvic origin.

On the other hand, in acute tubal inflammation, or in acute exacerbations of chronic pelvic inflammation *there is a different history.* There is usually an antecedent history of gonorrhea, evidenced by urethritis or profuse leukorrheal discharge, or of recent labor or abortion. The muscular rigidity is less marked and less well defined, and tenderness may usually be elicited over both Morris's points. The temperature is apt to be higher, but the patient less ill than in appendiceal infection.

The leukocyte count may show a high total white, but unless there has been tubal rupture, with spreading peritonitis, the percentage of polymorphonuclears is relatively low. By vagino-abdominal examination the tube may usually be found as a thickened, sensitive mass, to the side of, or behind the uterus.

In the very early stages of acute salpingitis, before an extensive exudate is poured out, there may be no palpable mass, though there is *exquisite sensitiveness in the posterior and lateral fornices.* The uterus, however, is always exquisitely sensitive to motion, because of the parametritis and localized peritonitis, which coëxists in some degree in all tubal infections.

It would seem hardly possible for gallbladder inflammation to so simulate salpingitis as to make a differentiation necessary. When the gallbladder is involved, there is sudden abdominal pain referred to the

epigastrium and vomiting follows, which is more or less persistent, depending upon the degree of peritoneal irritation present. The temperature is not much elevated, except in empyema, in which instance it may reach 102-104.5° F. There is tension of the right rectus below the costal margin, and tenderness over Mayo-Robson's point. Finally, if we have the patient sit up and lean forward, and the fingers of one hand are pressed in *under the costal margin of the liver,* the patient will, on taking a deep inspiration, *complain of exquisite tenderness.*

Nephrolithiasis and ureterolithiasis may be excluded by examination of the urine, catheterization of the ureters, marking of the wax tipped ureteral bougie, and the X-ray examination of the kidney and ureter.

Physical Signs of Pyosalpinx.—In pyosalpinx the tube forms a tumor which varies in size from that of the finger (8 x 6 x 2 cm.) to that of the fist (28 to 30 cm. in circumference). Because of its weight and primary downward displacement, it is usually situated behind and beside, rarely in front, of the uterus. It may be sausageshaped, lying obliquely in the *cul de sac* behind and, depending upon its size, displacing the uterus forward, upward, or to one side. It may fluctuate or be cystic in character, or it may be as dense as a fibroid, or the pelvis may be partly or wholly filled with a hard board-like exudative mass, which has none of the characteristic signs of a tubal tumor. This is especially true during acute exacerbations of subacute or chronic pelvic inflammation. The uterus is enlarged, more or less firmly fixed and displaced, upward, downward or laterally, depending upon the size and location of the mass, or the tube and uterus may be so matted together that neither is distinguishable in the general pelvic mass.

Recto-abdominal or vagino-abdominal examination, particularly under surgical anesthesia, is useful in detecting and mapping out the tumor and distinguishing it from the contiguous organs, to which it is always adherent.

Differential Diagnosis of Pyosalpinx.—We have to distinguish a pyosalpinx from appendicular abscess, parametritic abscess, fecal tumors, tubal pregnancy, hydro- and hematosalpinx, and ovarian tumors.

Ovarian tumors are generally more nearly globular and not so closely connected with the uterus. Under anesthesia there is usually a sharp line of separation which can be made out between the cyst and the uterus. Large ovarian tumors are more readily recognized by their size, consistency, and absence of inflammatory signs.

Tubal pregnancy, as a rule, can be diagnosticated by its typical history and the absence of an exudative mass.

Fecal tumors are recognized by the "pitting" which can be made in

the mass, on pressure by the finger, through the vaginal wall or by rectal exploration and by enemata.

A parametritic abscess usually displaces the uterus upward and to one side, while pyosalpinx displaces forward and to one side. Parametric abscess always follows labor, abortion, or intra-uterine instrumentation, and is so intimately blended with the uterus, that the lateral fornix, owing

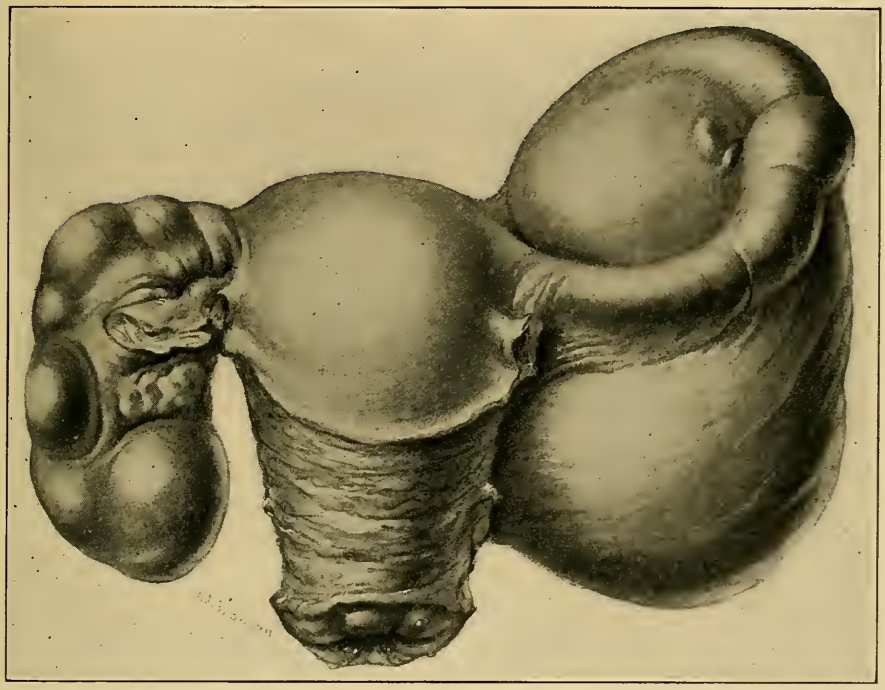

FIG. 47.—A PYOSALPINX ON ONE SIDE AND A TUBO-OVARIAN ABSCESS ON THE OTHER. FROM THE AUTHOR'S COLLECTION.

to the swelling in the parametrial connective tissue, is effaced and the cervix is flush with the vaginal vault.

Hydrosalpinx presents the greatest difficulty in differential diagnosis. In hydrosalpinx the pain, tenderness, and systemic disturbances are less marked than in pyosalpinx. The walls are thin and more fluctuant, and the tumor may even be so flaccid as not to be appreciable to the bimanual touch.

Hematosalpinx, the result of tubal pregnancy, is distinguished by its abrupt development, the characteristic metrorrhagia, and the physical signs. In acute inflammatory conditions within the pelvis satisfactory bimanual examination is often impossible without general surgical anesthesia, owing to the exquisite pain produced by the manipulation, and

examination at this time almost invariably increases the inflammatory reaction. Even under the most favorable circumstances a definite differential diagnosis is not always possible.

Appendicular abscess is usually suggested, if a careful history is noted, but when it has formed in conjunction with a pelvic peritonitis, adhesions may make the diagnosis more difficult. With appendicular abscess, on abdominal palpation, we find the uterus to be free and, for the most part, insensitive and freely movable. Recto-abdominal examination may show the abscess to be situated close to the pelvic brim pos-

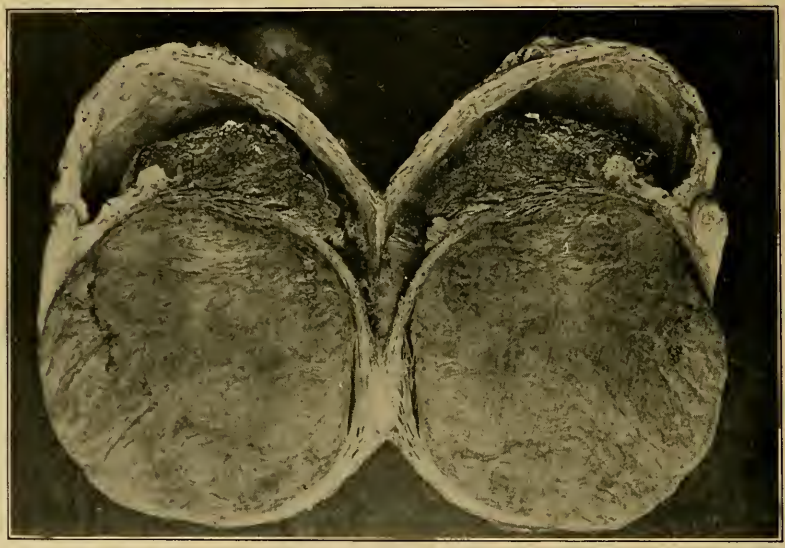

Fig. 48.—Acute Red Degeneration of a Myoma Complicating Pregnancy. Sudden Onset, Acute Pelvic and Abdominal Pain and Fever Rise in Pulse and Leukocytosis, with Rapid Increase in Size of Tumor Mass, which Became Exquisitely Sensitive, Simulating an Acute Abdomen.

teriorly. Occasionally a point of induration is found over the abscess area, and this may point to its true location.

Prognosis.—Probably in no other pelvic condition is the prognosis so dependent on an intelligent appreciation of the life history of the infecting organism and the pathology which it produces, as in inflammation of the fallopian tubes. In pure gonorrheal infection of the tube, time will, in a large majority of the cases, allow a complete recuperation of the tube. Mixed infections are not so favorable in their end results, for pyosalpinx is almost invariably the terminal condition of a mixed infection, while hydrosalpinx is a terminal result of streptococcic infection, reaching the tube by way of the parametrium and peritoneum.

Both pyosalpinx and hydrosalpinx are terminal conditions, the fin-

ished products of the inflammation, and, unless manifestations are excited by further bacterial invasion, as, for instance, from the intestine or through the uterus by untimely instrumentation near a menstrual period, these conditions may remain quiescent for a long period of time.

Purulent salpingitis terminates most frequently in pyosalpinx, but occasionally the abdominal ostium does not become sealed, and leakage of the tubal contents may escape into the peritoneal cavity. This in turn occasions recurrent attacks of pelvic peritonitis, which is relatively common in all chronic tubal inflammations, for it seldom happens that both ostia are completely closed, especially in the early stages. Clinically, we are apt to find one tube with its ostium completely closed, while the other is adherent by its fimbriae to some adjacent viscus, a situation which allows the escape of its infected contents under certain favorable conditions.

Gonorrheal salpingitis is seldom dangerous to life, for there is a definite tendency for the inflammatory reaction to be confined to areas within the true pelvis. On the other hand, *streptococcic salpingitis has a definite primary mortality,* and, owing to its longevity, greater virulence, and stronger tendency to extend beyond the confines of the true pelvis, one can never tell when a streptococcic infection will relight and prove fatal.

Rupture of a pyosalpinx may take place into the peritoneal cavity, producing a fatal spreading peritonitis, or it may rupture into and be walled off in the *cul de sac* of Douglas, where it causes an active reaction, which may be entirely confined within the pelvis and result in a pelvic abscess. In rupture into the peritoneal cavity with ascending peritonitis, sudden severe abdominal pain, high temperature, and a high leukocyte count, with a polymorphonuclear percentage of over ninety, are evidences of the virulence and severity of the extension; or it may rupture into the bladder and discharge its contents through that organ as a pyuria, or into the rectum or vagina, or even become adherent to the parietal peritoneum and extend through the abdominal wall or point in the region of the inguinal canal. These ruptures into adjacent viscera almost invariably result in the formation of permanent fistulae; consequently the relief caused by the evacuation of the purulent contents is only temporary, mixed infection occurs, and a reaccumulation follows. These pus sacs may intermittently empty themselves through the fistula during the life of the patient. Such patients become chronically septic and consequently are poor subjects for radical operations. Owing to the inflammatory reactions excited in the peritoneal covering of the intestines and parietal peritoneum, intestinal obstruction may complicate

these old cases of tubal inflammation. When once the intestine has become adherent to the tubal mass, the patient becomes a constant sufferer from intestinal disturbances, which may vary from simple gaseous distention to complete obstruction.

The pus of a gonorrheal or of a pyogenic organism, confined in the tube, may become sterile in from six to eight weeks, or may continue to be infective for a longer time, but rarely if ever for longer than three months.

As we have already stated, the two principal infective agents in tubal inflammations are the gonococcus and streptococcus. These organisms differ widely in the persistence of their virulence, but may usually be differentiated clinically, by the history of the infection and the location of the resulting lesions.

Gonorrheal infection commonly extends along the uterine mucosa to the tube, thence to the ovary and peritoneal cavity. Therefore, the characteristic lesion which we have found has been a suppurative salpingitis, a pyosalpinx or hydrosalpinx, with or without a complicating ovaritis and peritonitis, as the results of these inflammations.

Time effects many symptomatic cures in gonorrheal infection of the tubes, for we have seen many pelvic masses disappear, uteri become movable, and the adnexae insensitive. The tube may, under proper management, regenerate sufficiently to allow the passage of the ovum. A recorded number of intra-uterine pregnancies, and numberless ectopics, are evidence of this complete or partial regeneration.

Streptococcic exudates follow infections due to miscarriage, labor or intra-uterine instrumentation. *The streptococcic lesion is almost invariably in the parametrium.* From the port of entry such an infection passes through the parametrial cellular tissues by way of the lymphatics, and by contiguity of tissue to the peritoneum of the tube. When infection passes through these structures, it always causes a more or less sharp reaction, which produces an exudate, and hence we may state that in streptococcic, tubal infection *there is always an associated parametritis.*

This chronic parametrial mass is in the broad ligament, located low down beside the cervix, and blends with the uterine wall, making the lower part of the uterus appear broader and more fixed. In considering the operative prognosis and management of these streptococcic tubal infections complicated by parametrial exudate, it must be remembered that the streptococcus may lie dormant for even ten or fifteen years, and then light up after surgical intervention and produce a fatal sepsis.

We have had four such cases, where the streptococcus lay buried in a parametrial exudate (according to the history) for five, eight, ten

and fifteen years respectively, and was liberated into the circulation; in one by a spontaneous labor, in another by a curettage for hemorrhage, and in two by a simple abdominal section for chronic tubal inflammation. All four cases subsequently died of a streptococcic bacteriemia.

In the author's study of the cause of sterility, he was amazed to find the large number of women who were sterile because of chronic tubal inflammation, which is not appreciable even to the expert on bimanual examination. It has been our rule for several years never to treat a case of sterility without having the condition of the male member of the partnership checked up by a competent urologist, not only as to his potency but as to his previous or present infections. This study has shown that a large number of men marry who are potentially infective; that is, who have infective pyogenic cocci in their prostatic secretions, even when no true gonococci can be found. Furthermore, diplococci of an extracellular type and staphylococci have frequently been demonstrated.

From these facts, we believe that a man who marries with staphylococci in his prostate can and does infect his wife by setting up an infective endocervicitis, which of itself does no harm except to produce a constant mucopurulent discharge. If we dilate such a cervix, or curet the uterus, the cervical infection is passed on into the uterus and tubal inflammation follows. Clinically, we have proven the truth of this statement in over two hundred abdominal sections done for sterility. These tubes always present the same picture, i.e., they are distorted and nodulated, with thin peritubal adhesions. Sterility and chronic invalidism are often the results of tubal inflammation.

In tuberculous infection of the tubes the process may extend to the peritoneum, with a resulting pelvic peritonitis, or may become generalized. Spontaneous recovery is possible, as in other forms of tuberculosis.

The writer has seen inoperable tuberculous salpingitis become quiescent, and remain so for many years, only to relight when the patient's resistance was lowered by some other intercurrent disease.

The operative prognosis in septic and gonorrheal cases of tubal inflammation, when the operation is done after all of the acute symptoms have subsided, is good, and if the proper preoperative care is given to these women, the mortality should not exceed one per cent.

Treatment.—The treatment of salpingitis must be considered under the following headings:

1. Acute salpingitis.
2. Acute exacerbations of chronic salpingitis.
3. Chronic salpingitis, palliative, conservative, radical.

Treatment of Acute Salpingitis.—*No case of acute salpingitis needs operation.* The management during the acute stage consists of rest in bed, with the patient in the Fowler position, to favor postural drainage, and the employment of hot or cold applications over the lower abdomen. We have found that a hot antiphlogistine poultice spread on cotton batting and applied over both lower quadrants, with a hot water bag placed over the poultice to maintain the heat, and both the poultice and the bag held in position with a snug many tailed binder, serves the purpose of supplying heat and moisture to the parts, and relieves the pain of inflammatory reaction.

Fig. 49.—Drawing from Operation Showing How Sigmoid and Omentum Close off the General Peritoneal Cavity in Pelvic Inflammation.

Other patients find that cold is more agreeable and has a better anesthetic effect than heat. If this is found to be the case, an ice bag or an ice coil may be applied over the affected side or across the entire lower abdomen.

Salpingitis tends to spread by extension to the peritoneum, and *peritonitis is spread by peristalsis;* hence we have learned that postural drainage, which is accomplished by the use of the Fowler elevated trunk posture, tends to control the upward extension of the infection and to keep the inflammatory reaction within the true pelvis, by allowing the sigmoid and omentum to close off the pelvic viscera from the general peritoneal cavity (Fig. 49). *No cathartic should be used.* Enemata may be employed to empty the lower bowel, but these should not be larger than eight to sixteen ounces, as it is desirable to limit their effect to the terminal portion of the large bowel and *not excite intestinal peristalsis.*

The pain may be controlled with some form of opium. It is well to administer this in small and repeated doses, as it takes less anodyne to control pain when a small dose of the drug is repeated at definite intervals than when a larger dose is given, only when the patient is

suffering severely. Besides relieving pain, opium and its derivatives diminish the intestinal peristalsis and so tend to prevent the upward extension of the associated peritonitis.

Time does much toward allowing the pelvic organs to reassume their normal functions. "Watchful waiting" is, therefore, the slogan in acute salpingitis.

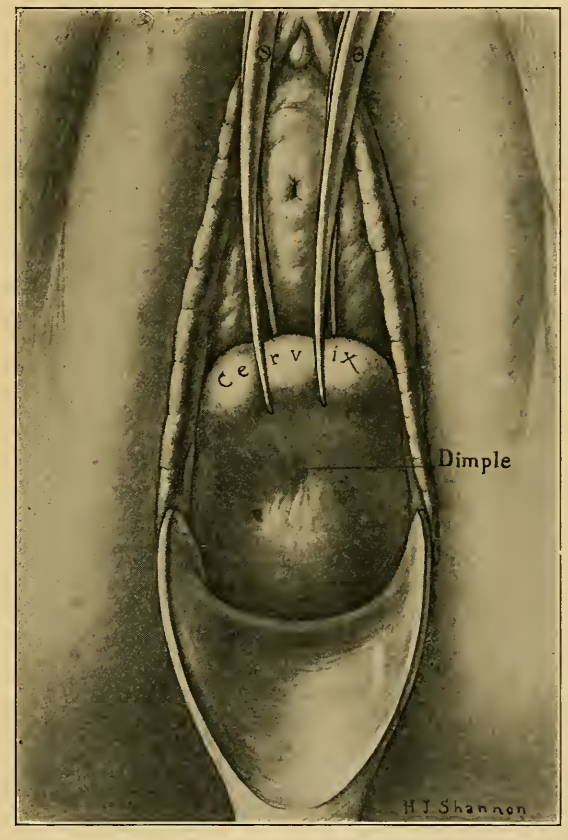

Fig. 50.—Technic of Posterior Vaginal Section, Showing Point of Incision.

After all temperature has subsided and the local symptoms have become subacute, *hot vaginal douches of 120° F.* may be given twice daily. These douches do not need to contain any medicant, but should be of such quantity that it takes from fifteen to twenty minutes to administer, i.e., four to eight quarts. The douche should be given with the reservoir at low elevation. This supplies prolonged heat without producing trauma.

Counterirritation to the vaginal vault by the application of the tincture of iodin or, as suggested by Ill, the employment of cantharides in collodion, applied to the posterior fornix over the exudate, may aid reso-

lution. When the exudate is large, dry heat, such as can be supplied with the Robinson "thermolite" lamp or the Gellhorn baker, hastens the absorption of the mass.

It will be seen, therefore, that the *fundamentals in the treatment of acute salpingitis are rest, posture, opium, inhibition of peristalsis (intestinal rest), and time.*

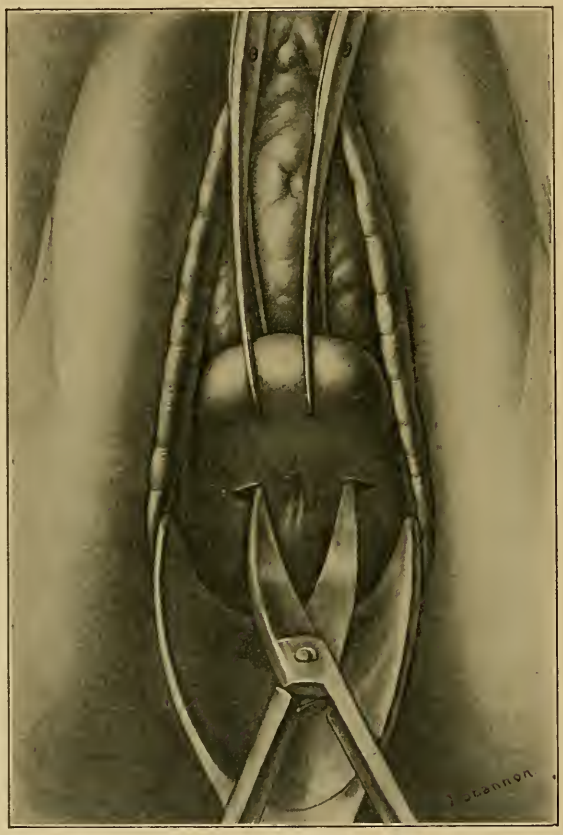

FIG. 51.—TECHNIC OF POSTERIOR VAGINAL SECTION: MAKING THE INCISION THROUGH THE VAGINAL MUCOSA.

Under this general plan of treatment, more or less complete resolution takes place. Unfortunately, in a few cases, instead of the protective exudate being absorbed, an abscess forms in the *cul de sac* of Douglas. When this occurs, the acute peritoneal symptoms subside, the temperature becomes remittent or intermittent, the polymorphonuclear percentage falls, and the patient usually complains of pressure symptoms in the pelvis, as evidenced by rectal and vesical tenesmus.

The vaginal findings show a bulging and soft spot in the posterior fornix, while the uterus is displaced upward behind the pubis. When

the diagnosis of pelvic abscess is made, its contents may be evacuated by posterior vaginal section.

Vaginal section is also of value in acute spreading peritonitis of pelvic origin due to abortion or tubal leakage. Our index for surgical interference in these cases has been the occurrence of sudden abdominal pain, which is not relieved by the moderate use of morphin, abdominal tension,

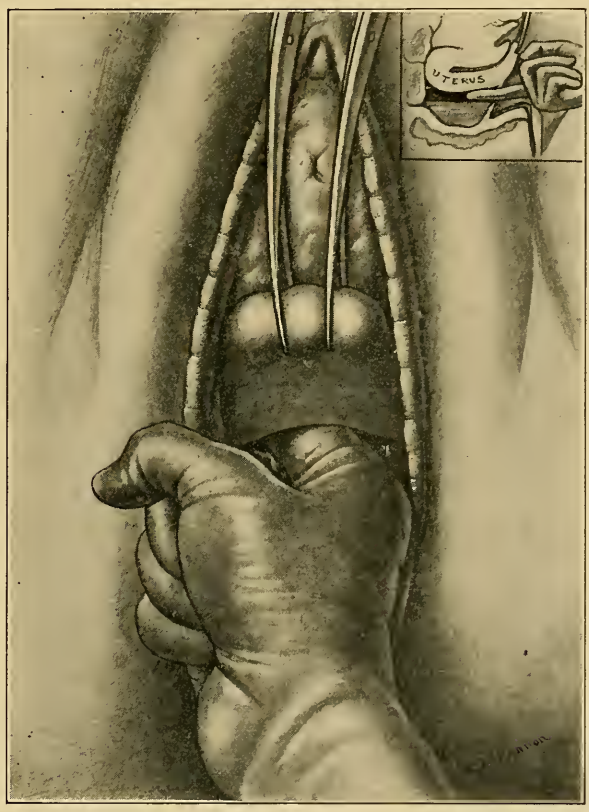

FIG. 52.—THE SCISSORS ARE THEN DISCARDED AND THE FINGER IS INTRODUCED THROUGH THE INCISION IN THE VAGINAL MUCOSA.

tenderness, high temperature, a high leukocyte count, and a polymorphonuclear percentage of over ninety. Furthermore, posterior colpotomy and drainage is valuable as a palliative measure in the treatment of pus tubes and infected ovarian cysts which are located in the true pelvis, when such a patient shows signs of septic intoxication, as evidenced by persistent temperature above 101° F., a leukocyte count of between 15,000 and 22,000, with a polymorphonuclear percentage of eighty-five or above. Such patients improve rapidly when the septic focus is evacuated. At a later time radical operation may be safely done.

The Technic of Vaginal Section (posterior colpotomy) for the *cul de sac* drainage of pelvic abscesses, acute spreading peritonitis of pelvic origin, large pus tubes, and infected ovarian cysts, is as follows:

After properly cleansing the vulva and the vagina, with the patient in the lithotomy position, a large operating vaginal speculum is introduced, to retract the posterior vaginal wall and expose the cervix (Fig.

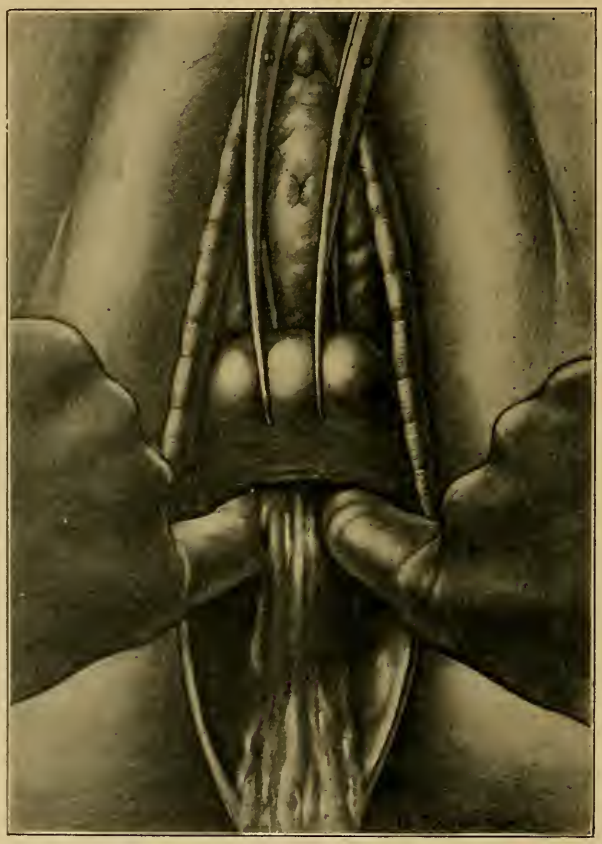

Fig. 53.—Widening the Incision with the Fingers.

50). The posterior lip of the cervix is caught with two Skene double tenacula and drawn downward and forward. This procedure exposes the *posterior uterovaginal junction* and develops a depression or dimple between the uterosacral ligaments at their uterine attachment (Fig. 51). At this point, with a pair of long sharp pointed scissors curved on the flat, a transverse incision is made through the vaginal mucosa. The incision should be about an inch and a half to two inches in length. The scissors may now be discarded, the speculum removed, and the finger

introduced through the incision in the vaginal mucosa; and, as the cervix is drawn forward, the finger is kept close to the posterior uterine wall, it is then pushed through the cellular tissue and peritoneum until the *cul de sac* is entered and the contained pus liberated. The index finger of each hand is now passed into the incision, and the wound spread to admit three fingers.

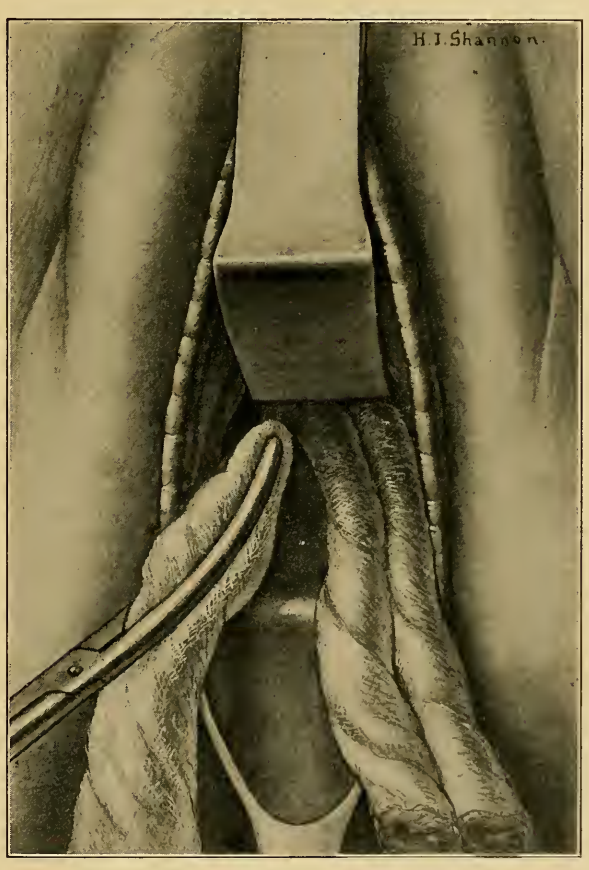

Fig. 54.—Gauze Rolls Placed in the Cul de Sac for Drainage Are Preferable to a Rubber Tube.

The pelvic cavity is next explored for secondary pus pockets and, if any are found, these should be evacuated. If an infected cyst or large pus tube presents itself in the colpotomy wound, it should be carefully punctured with the scissors and the opening enlarged by the finger. A long Sims speculum is now introduced into the colpotomy wound, to hold back the posterior vaginal wall, while a Deaver retractor, placed anteriorly in the incision, holds the uterus forward. With these retractors properly placed, the cyst or tube cavity may be exposed and a

T-shaped rubber tube of large caliber placed in the cyst for drainage, while the *cul de sac* is filled with twisted, washed out iodoform gauze drains, two by twelve inches, arranged across the pelvis (Fig. 54). We usually place five of these twisted drains in the pelvis and leave them in place for eight to ten days, removing the middle one first. This insures a large patent opening in the *cul de sac* that will not become occluded before the cavity above has closed down.

It must be remembered that *drainage depends on the size of the incision through which the drains emerge,* hence the necessity of a large opening in the vaginal vault. By following this technic, we have never had to repeat our drainage procedure. Irrigation of the abscess cavity is never used, for the dangers of trauma and of the dissemination of infective material beyond the pelvis counterbalance any theoretical advantage. The elevated trunk posture secures proper gravity drainage, and after the drains are removed, the patient is instructed to lie upon her abdomen for several hours daily, to ensure emptying the vagina.

Operative intervention by the abdominal route is generally contraindicated during the early stage of any acute inflammation; first, because the patient may recover under palliative treatment; second, because collections of pus in the pelvis become less virulent or sterile, if sufficient time is allowed for the process to become quiescent. Diffuse spreading peritonitis is the exception to this rule, and, when present, free drainage through a stab wound incision is demanded.

Occasionally, in these acute pelvic inflammations, abdominal distention, tympany, and vomiting become prominent factors. Starvation and lavage will control the vomiting and the distention in the upper intestinal tract, while proctoclysis, after the method described by Harris, will furnish the necessary fluids for the patient, and promote the escape of gas from the large bowel.

The Harris drip is constructed from an ordinary tin or agate douche can with three to four feet of soft rubber tubing, to the distal end of which an ordinary rectal tube is attached. The can is placed at an elevation which exactly corresponds to the height of the pubis of the patient. After being half filled with a five per cent glucose solution, at a temperature of 105° F., a small quantity of the fluid is allowed to escape through the rectal tube, and, while it is still running, the tube is inserted into the anus; the fluid promptly seeks its level, and the solution in the bowel will be at the same height as the solution in the agate reservoir. By keeping this constant column of fluid in the rectum and sigmoid under low pressure, large quantities are absorbed by the colon and little or no irritation is produced by the contained fluid. Both feces

and gas are passed through the tube back into the containing reservoir. The temperature of the solution may be maintained by placing a lighted incandescent (carbon) bulb in the douche can.

Before any operative procedure is justifiable, all acute symptoms must have subsided, and the morning and evening temperatures must be normal for a period of at least three weeks, all exudate must have been absorbed, or if some still remains, it must be hard and insensitive. Pelvic examination will not, if these conditions are fulfilled, excite an exacerbation of temperature or an increase in the leukocyte count. *The leukocyte count should never be over eleven thousand nor the polymorphonuclear percentage over seventy-five at the time of making an abdominal section for tubal inflammatory disease.* It is our custom to test these cases by making a complete blood count and then to subject the patient to a thorough bimanual pelvic examination. If this examination does not free any toxins, the temperature and leukocyte count will not be raised and operation may be safely done.

Treatment of Chronic Salpingitis may be palliative, conservative or radical. As we have stated before, time effects a symptomatic cure in a large number of tubal inflammations. This is especially true when the gonococcus has been demonstrated to be the only infective agent present. The tube is often able to recuperate completely and have the patency of its lumen completely restored; therefore we feel that there is considerable virtue in the palliative management of these subacute and chronic inflammations.

Palliative.—The treatment consists in the maintenance of the woman's general health by tonics, fresh air, proper rest, and marital continence. The regulation of the intestinal tract should be accomplished by the use of systematic abdominal exercise, proper diet, and the administration of mineral oil and enemata. When cathartics are used, only those which exert their action on the rectum and lower sigmoid should be employed. On general principles, it is better to avoid the use of cathartics entirely.

The associated cervicitis, endocervicitis, and posterior parametritis which causes the leukorrhea, the backache, the dyspareunia, and the premenstrual pain, may be relieved by proper local treatment. This consists (1) in marital abstinence; and (2) in the absorption of the cellular exudate in the surrounding ligaments.

In the first instance, we may or may not succeed, depending entirely upon the proclivities of the contracting parties. To accomplish the second result, hot vaginal douches with two or four quarts of water at a temperature of 120° F., taken before retiring, with the patient in

the recumbent posture and the bag at low elevation, tends to change the pelvic circulation and relieve the circulatory stasis, and thus improve the pelvic soreness.

With the patient in the knee-chest position, counter irritation of the fornices and local disinfection of the cervical area by an application of strong Churchill's tincture of iodin over the cervix, cervical canal, and vaginal fornices, tends to relieve the local pain.

In the author's experience, it has not been necessary in these chronic cases to use more active counter irritants, such as cantharides collodion. Better results have been obtained by the systematic employment of the "Gellhorn baker," applying the dry heat over the lower abdomen at a temp rature varying from 140° to 250° F.

Conscientious persistence in maintaining the patient's general health, regulation of the bowels, douches, iodin, heat and, above all, *time* have relieved many of these patients of all of their subjective symptoms.

It is most important that the *patient* who is the subject of chronic pelvic inflammation *learn to properly care for herself* just prior to each menstrual period, as it is at this time that exacerbations are most likely to occur. These women should be instructed to rest in bed previous to and during the period, to abstain from meat for four or five days before the onset of the bleeding, and finally to be sure that the lower bowel is emptied by an enema six to twelve hours before the menstruation begins.

The Operative Treatment of Chronic Salpingitis includes (1) salpingostomy, or the partial resection of one or both tubes, and (2) salpingectomy, or ablation of one or both tubes with a retention of the uterus and one or both ovaries, or part of the uterus, with one or both ovaries to maintain the menstrual function.

Salpingostomy has a limited field. This operation is only permissible in young women, in an occluded tube with its free portion and ampulla distended, but without persistent active inflammation; as, for instance, in certain cases of terminated hydrosalpinx which intermittently discharge their contents through the uterine cavity, and in tubal pregnancy operated upon before rupture. In other words, salpingostomy may be done where it can be demonstrated that the interstitial portion of the tube is patent. It is never permissible in a gonorrheal or tubercular infection, even where there is no pus, as the tube's subsequent behavior has demonstrated that conservative surgery has no value.

Pregnancies have occurred where this conservative procedure has been done, but the ultimate termination is always questionable, as we have no means of knowing whether or not any bacteria persist in the tube walls. We have reported the case of one woman who has had

three children subsequent to a phimosis operation on the tubes, as well as three cases of intra-uterine pregnancy in which the terminal portions of both tubes were resected. In addition to this, we have records of seven ectopic pregnancies occurring in the proximal portion of resected tubes.

Notwithstanding the many recorded failures in the hands of reliable operators, there are those who still believe in this form of conservatism. However, we are convinced that, due to the pathological findings in practically all of these pelvic infections, whether of gonorrheal or streptococcic origin, it is very doubtful whether conservatism accomplishes sufficient in the end to compensate for the risk that accompanies it. After all, *conservatism may eventually become radicalism.*

If a woman is chronically invalided by the retention of an infected tube or tubes following conservative salpingostomy, what chances has she of becoming pregnant, and if conception takes place, what chances has she of carrying to term? Furthermore, in case no conception follows, but the symptoms for which she originally suffered are constant and unabating, what state will her mental attitude assume when a second operation is advised as her only alternative for health? Such questions cannot be answered with any degree of certainty. However, it may be assumed with certainty, that a woman who is healthy and free of symptoms (pain, leukorrhea, etc.) and who menstruates regularly, which to her always means a possibility of conception, is far happier and contented than one who has symptoms and menstruates irregularly and finally becomes convinced that she cannot become pregnant because she is sick. Expectation may therefore produce happiness or misery, depending upon the physical and mental state of the expectant.

Technic of the Procedure.—With the patient properly prepared and in the Trendelenburg posture, through a free abdominal incision the tube is freed from its parietal and visceral adhesions, and it is brought up into the abdominal wound, after isolating and inspecting the adjacent ovary. The peritoneum is now carefully walled off with wet gauze laparotomy sponges and the tubal tumor incised along its dorsum. This allows the escape of the contained fluid and permits inspection of the lining membrane. A fine ear probe or a number one filiform bougie may now be passed through the isthmic and interstitial portions of the tube into the uterus. If the lumen is patent, this can be replaced by a single strand of "oo" chromicized catgut. When the patency is thus assured, we evert the mucosa around the trumpet-like end by suturing it with very fine catgut to the peritubal peritoneum. In this way we have a large funnel shaped opening, without raw edges, for the receipt of the ovum.

If sufficient care is taken in this eversion and all raw surfaces are completely covered, some success may be looked for. To increase the chance of pregnancy, the ovary should be fastened near the open end of the tube, great care being taken to avoid passing any suture through the protective tunic.

Partial resection of the tubes has little or no clinical value as a conservative operation, except to allow an opportunity for the arrest in transit of the fecundated ovum. Tweedy and his assistant have each recently reported cases of ectopic pregnancy occurring in resected tubes. Two of our interstitial pregnancies have occurred in the stumps of tubes which had been previously resected and ligated. It must always be remembered that the spermatozoön can pass through a smaller lumen than

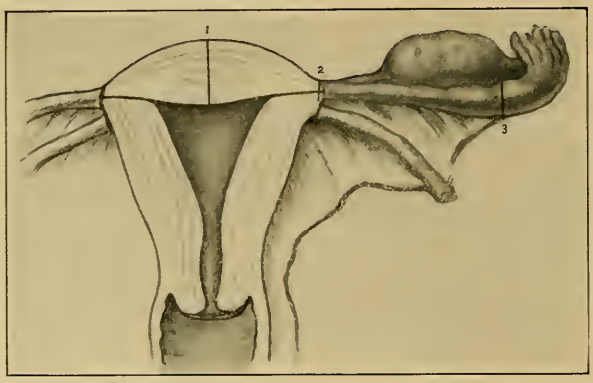

Fig. 55.—Serial Sections Were Taken from Areas Marked 1, 2 and 3.

the ovum, hence the danger of ectopic pregnancy in these attempts at conservatism.

Complete ablation of the tube is the common procedure in chronic tubal inflammations which demand operation. To our mind, if the infective process is one of long standing, the uterine tissue about the interstitial portion of the tube is always involved to a greater or less degree. We have shown by serial sections through this region that inflammatory tissue is found at a considerable distance from the infected focus, and that unless this tissue is widely removed the woman carries with her, through life, an infected hyperplastic uterus.

Furthermore, in this same class of cases, the cervix should also be removed, for, like the fundal area, it also is a constant source of infection. (The reader is referred to the chapter on Endocervicitis, that he may appreciate the significance of this statement.)

Leukorrhea would be cured by this procedure, while, if the cervix were not removed, leukorrhea would persist.

Technic of Salpingectomy.—With the abdomen open and the pelvis exposed and walled off by placing a moist gauze laparectomy pad across the brim, the tubes are freed from adhesions, the ovaries isolated and inspected, and the tubes brought up into the operative field by traction with a rubber covered ring forceps. Beginning at the distal end, the vessels of the mesosalpinx are isolated and tied separately and the tube

FIG. 56.—CELL REACTION IN TUBAL SECTION FROM AREA 3 SHOWING SMALL ROUND MUCOSA.

cut free of its mesosalpinx (Fig. 59) as we go along. When the cornu of the uterus is reached, the two sheets of the broad ligament are separated with Mayo scissors and the fundal branch of the uterus, after it leaves the utero-ovarian anastomosis, isolated and tied. This allows us to cone out and remove the interstitial portion of the tube without bleeding, and close the cornu with sutures. Unless this portion of the tube is removed, a focal infection lurks in the stump and a local peri-

tonitis at this point follows. This favors omental and intestinal adhesions and thus, by increasing the circulation, allows a persistence of the uterine symptoms.

It was formerly supposed that no fluid accumulation could occur in the tube until both the uterine and abdominal ends were sealed, but on making serial sections through the isthmic and interstitial portions of the tube in a large series of cases, we have found that the lumen of the uterine end never closes, and that *the apparent occlusion* in the isthmic and interstitial portions, which allows tubal distention, is relative and

FIG. 57.—SECTION FROM AREA 2 SHOWING REACTION ABOUT AND REMOTE FROM INTERSTITIAL PORTION OF TUBE.

not actual. This may be explained by the fact that the folding, edema, and consequent swelling of the mucosa make the actual lumen so tortuous that intratubal pressure closes or practically closes the uterine end. If one recalls for a moment the anatomy of the interstitial portion of the tube, surrounded as it is with an inner circular muscular coat, which is continuous with the circular muscular coat of the uterus, one must recognize the fact that any inflammatory process of the mucosa must

excite an inflammatory reaction in the muscular structures immediately surrounding the mucous tube.

The muscular coat, when examined microscopically, is edematous and infiltrated with inflammatory tissue cells and plasma cells, and in mixed infections, particularly when the streptococcus is present, there may be found small, localized abscesses in the surrounding muscle tissue. This small, round, tissue cell infiltration results in an increase in the connective tissues within the uterine wall and thereby actually increases the size of the uterus. These metritic changes do not subside after the removal of the distal portion of the tubes, but may be found as infiltrated or fibrous nodules in the cornua, at subsequent operation or at autopsy.

Fig. 58.—Section from Area 1 Shows Inflammatory Reaction Remote from Tubal Entrance.

A knowledege of the persistence of this infection of the uterine muscle formerly tempted the French operators to do vaginal hysterectomy with double salpingo-oöphorectomy for chronic suppurative tubal infection; while, in this country, many of our more conservative surgeons, who acknowledge that a mixed infection of the tube can never be wholly cured without ablation of both tubes, and that the intractable leukorrhea which, under certain stimuli, regains its infective properties, and the increased amount of menstrual flow, which, together with the pelvic pain, make up the clinical syndrome, are all due to the enlargement and increased blood supply of the metritic uterus, and because of the persistence of these symptoms have advocated hysterosalpingectomy, with the retention of one or both ovaries. While we are agreed that the retention of an infected uterus is a menace to the

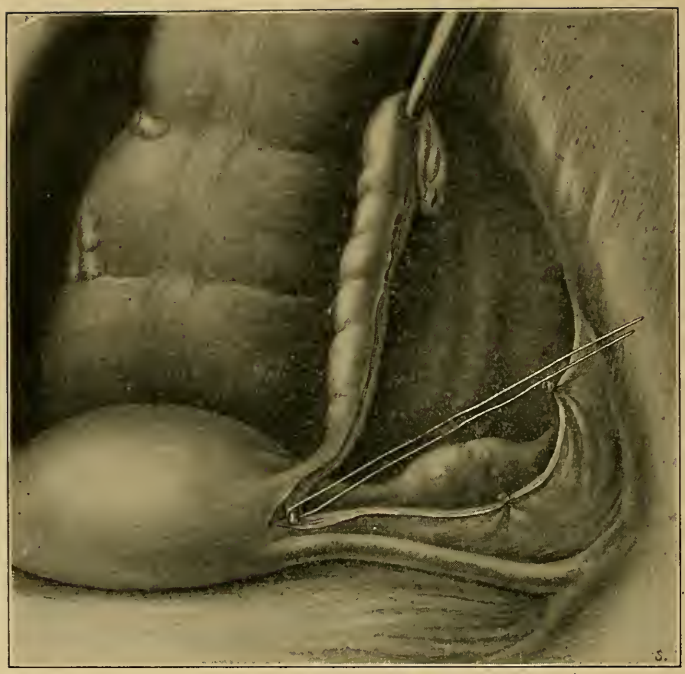

Fig. 59.—Freeing the Tube from its Mesosalpinx by Ligating the Individual Vessels and Not Interfering with the Ovarian Circulation.

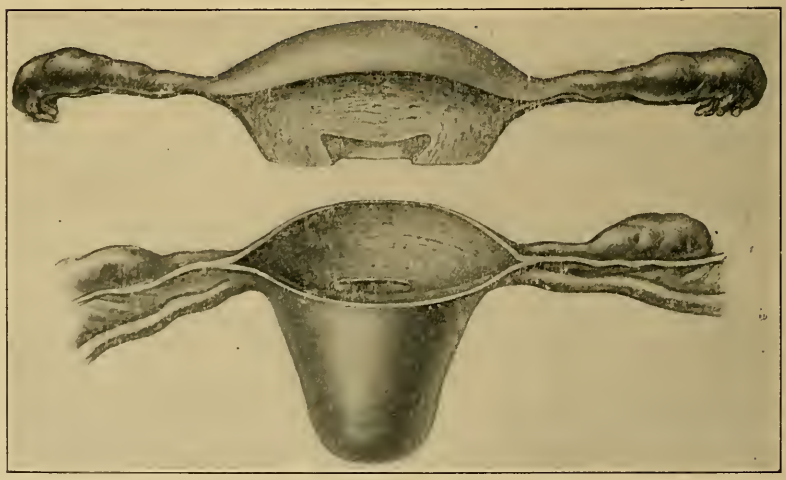

Fig. 60.—Showing the Extent of the Wedge Excised from Fundus and Body.

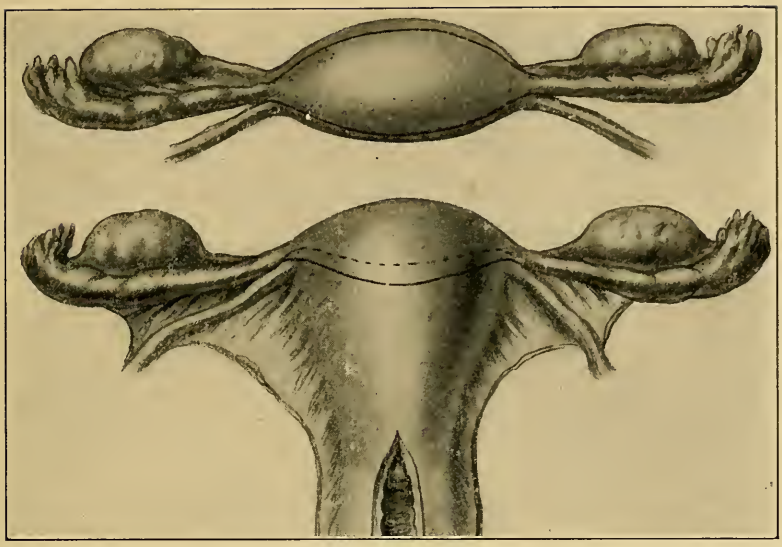

Fig. 61.—Showing Lines of Incision in Making a Partial Resection of the Uterus Including the "Pars Interstitialis."

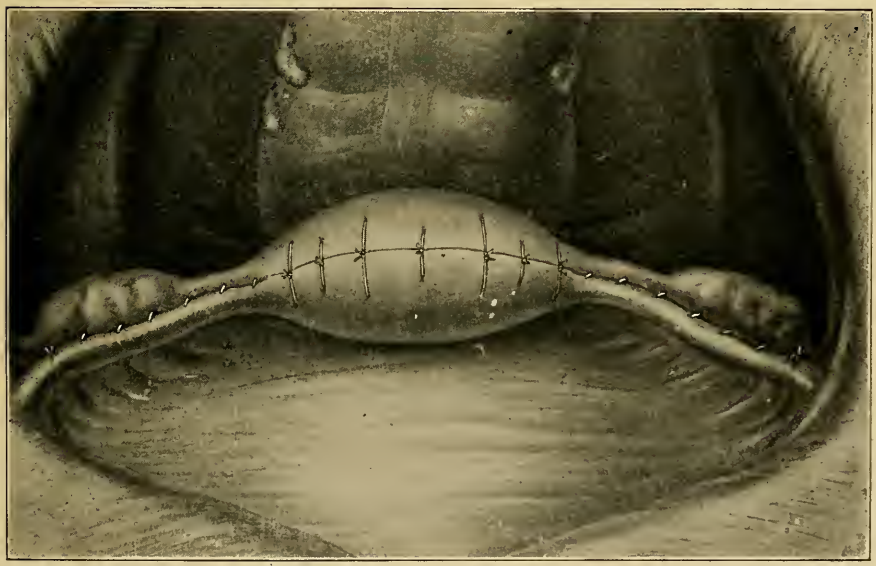

Fig. 62.—The Uterine Flaps are Then Brought Together with Interrupted Sutures.

woman's future health, our own feeling has been that, when so much care is taken to preserve the function of ovulation, there should be like care in preserving to the woman the function of menstruation, for ovulation without menstruation contributes little to the patient's well being, and furthermore, a study of serial sections through the uterus between the fundus and the internal os, show few, if any, of the pathological changes which have been demonstrated about the pars interstitialis.

It is *only in the cervical region, in Naboth's glands, in the columnar*

FIG. 63.—THE WOUND IS THEN PERITONEALIZED BY DETACHING THE BLADDER AND REFLECTING THE FLAP OVER THE SUTURE LINE—THE OVARIES ARE SUSPENDED BY SUTURING THEM TO ROUND LIGAMENTS.

epithelium of the cervical canal, in the fundal region, and in the structures surrounding the pars interstitialis, that there is a persistence of the inflammatory changes which have already been described.

Curtis has demonstrated that the interior of the uterus is capable of ridding and does rid itself of any trace of the infective process; hence, it must appeal to any one of any considerable experience that, in a given case of chronic specific infection, involving the cervix and the tubes, a cure cannot be effected except by the removal of these foci. The endocervicitis and cervicitis may be controlled by conical excision of the infected portion of the portio vaginalis, but the results of the tubal infection cannot be checked and its sequelae removed, without excision of the pars interstitialis and the structures immediately surrounding it. Consequently, *to preserve ovulation as well as the menstrual function in young*

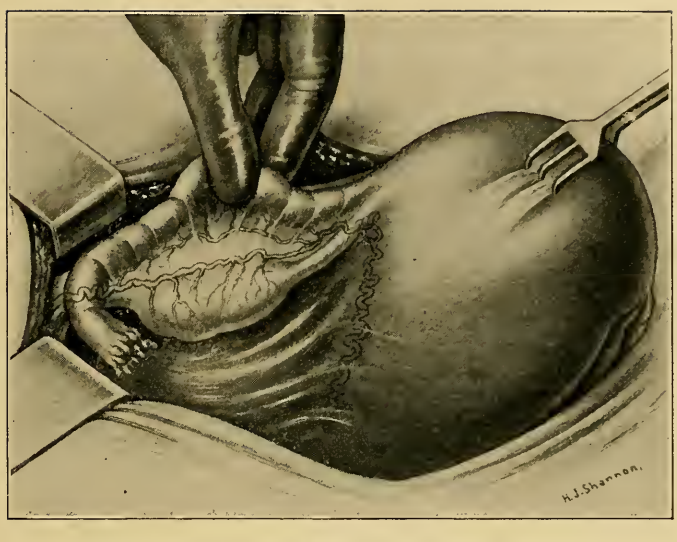

Fig. 64.—Showing the Interovarian Circulation.

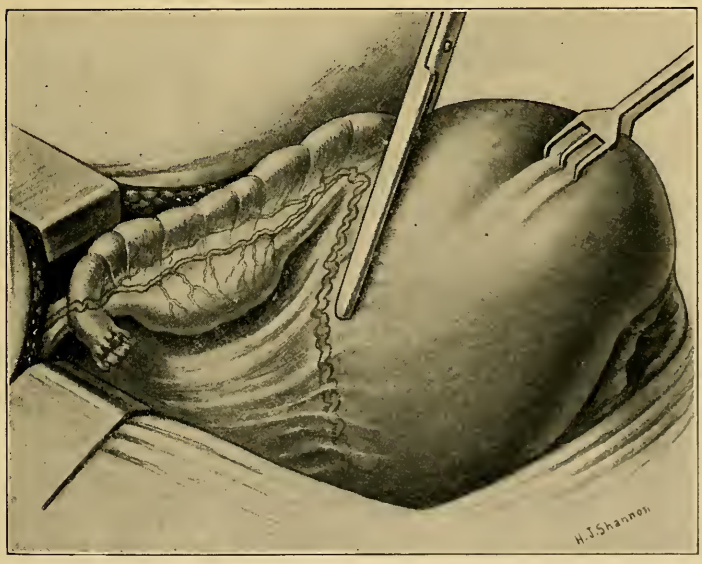

Fig. 65.—Conserving the Ovarian Circulation by Pushing the Utero-Ovarian Anastomosis off into the Folds of the Cross Ligament.

women suffering from chronic endocervical and tubal infection, it has been our custom to cure the leukorrhea by the radical excision of the infected area in the cervix, and follow this plastic procedure by the ablation of both tubes, with the resection of the infected fundus of the uterus, using a modified Bell-Beuttner technic. This leaves a sufficient amount of healthy uterine body, with its contained mucosa, to conserve the menstrual function and one or both ovaries to continue ovulation; for the ovaries are seldom grossly diseased if their tunica is intact.

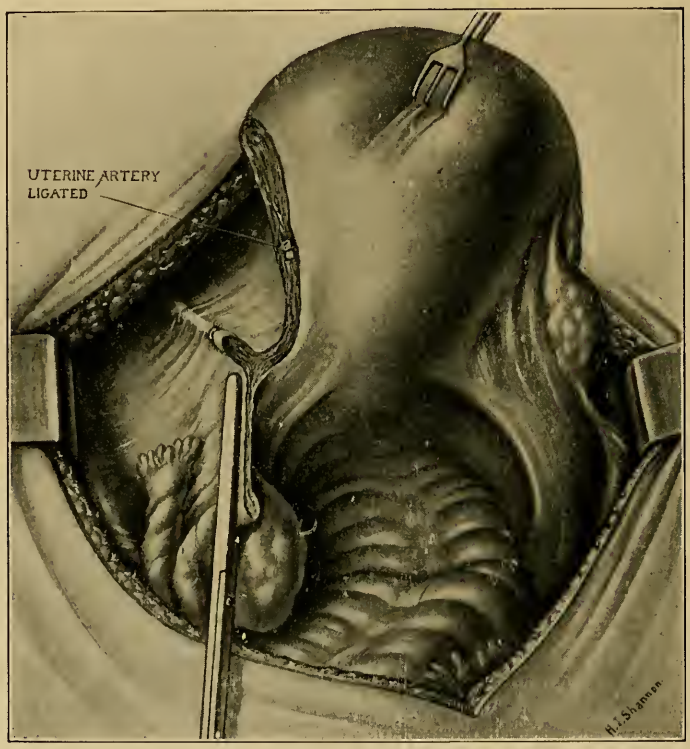

Fig. 66.—Uterine Artery Ligated—Mesosalpinx and Ovarian Ligament in Grasp of a Hemostat Forceps.

Removal of the tubes and resection of the upper segment of the uterus can be done without interfering with the ovarian circulation. Hence, the ovary may be retained with a greater degree of security, than if a hysterectomy is done and the uterine end of the anastomosis cut off. Furthermore, the functional life of the retained ovary or ovaries is longer if menstruation is conserved than if the uterus is removed. This, I believe, is due to the relief of the periodic premenstrual congestion by the uterine flow. The ovary is the analogue of the testicle, and we feel that *considerable risk is justified for the preservation of an ovary* and its cir-

culation. For it is well known that double oöphorectomy, particularly in a young woman, may convert the normal woman into a hopeless neurasthenic. In tubal disease the ovary is usually involved only because of its association with the infected tube. The lesion is a peri-ovaritis, rather than an ovaritis. It is an ovary in bad company, rather than a diseased ovary, and *its function can be preserved, provided its circulation is not interfered with.*

Menstruation exercises a therapeutic effect on health, which is not

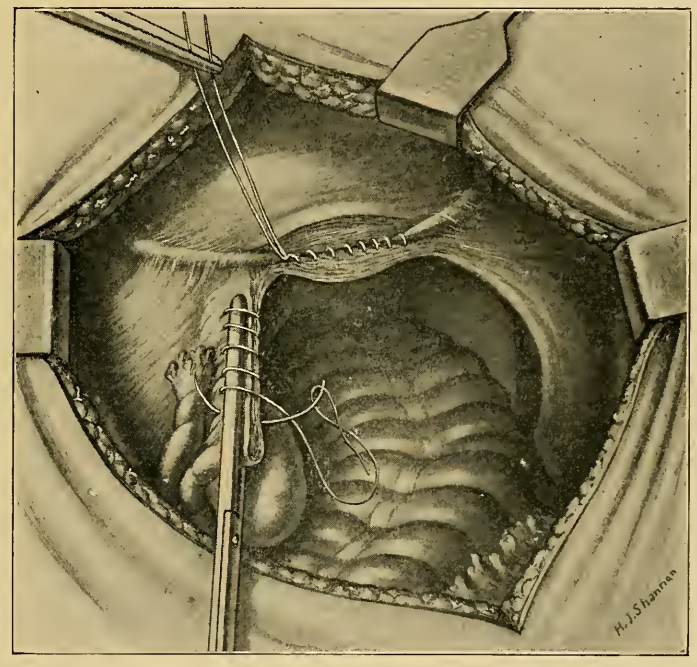

FIG. 67.—UTERUS REMOVED, ROUND LIGAMENTS SEWN INTO STUMP—A RUNNING SUTURE IS BEING PASSED AROUND THE HEMOSTAT.

only psychic, but actual, for the whole system in the female is planned for this periodic outflow. The uterine mucosa undergoes definite histologic changes throughout a definite cycle, which culminates in the menstrual flow. These cyclic changes are subconscious when proper correlation exists between the several governing internal secretions of the body; but when this correlation is disturbed by cystic changes in the ovary, which always result when its circulation is interfered with, definite symptoms are produced in the breasts, the tonsils, the thyroid gland, the genital spot in the nasal mucous membrane, and in the circulatory and nervous systems of the individual. Consequently, there can be no question as to the desirability of the conservation of the ovarian func-

tion, provided it is associated with the periodic overflow of menstrual blood. To have this, the ovary and the uterus must be comparatively healthy. As has been already stated, a healthy ovary or ovaries and a portion of healthy uterus can be conserved by the employment of the fundal resection.

Technic.—The success of the Bell-Beuttner procedure depends on the ligation of the fundal branch of the uterine artery, supplying the interstitial portion of the tube and uterine fundus. This is done, as in sal-

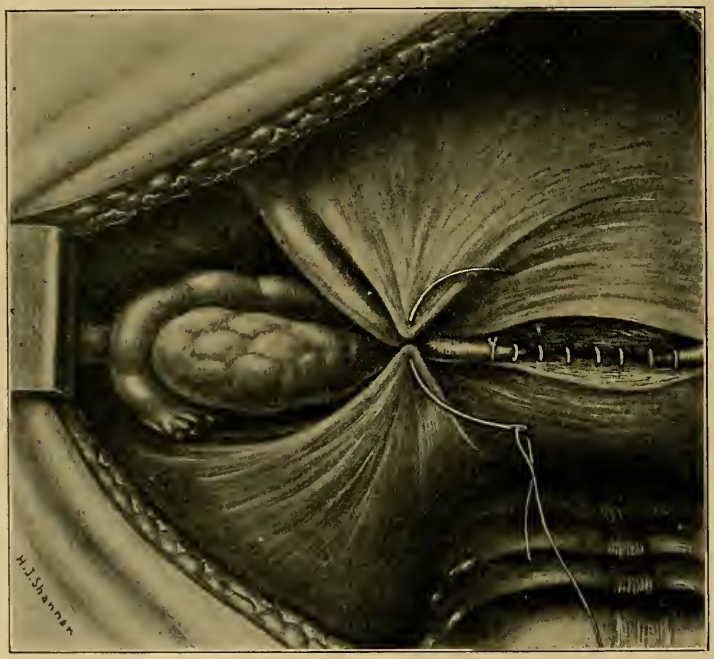

Fig. 68.—Conserved Ovary and Tube Suspended by Suture to the Round Ligament Supported by Peritoneal Pocket—This Method is Applicable Only when One Tube is Normal.

pingectomy, by separating the anterior and posterior sheets of the broad ligament and exposing the vessel just after it leaves the utero-ovarian anastomosis. With the bleeding controlled, a wedge shaped excision is made of the upper part of the body and fundus of the uterus (Fig. 60). The anterior incision begins just posterior to the insertion of the round ligament and runs across the front of the uterus to a corresponding point on the opposite side (Fig. 66). The posterior incision begins between the tubal insertion and the ovarian ligament on one side, and extends across the posterior surface of the uterus to the same point on the opposite side. The incision is wedge shaped and is made in such a manner that the entire fundal mucosa, with the pars interstitialis and

the surrounding tissues of both sides, is included in the excised mass of uterine tissue. The uterine flaps are then brought together with interrupted catgut sutures, superficial sutures being placed between the deeper ones. This wound is then peritonealized by detaching the bladder, as is done in panhysterectomy, and the peritoneal flap is reflected over the line of incision; this anteverts the uterus, beside practically making

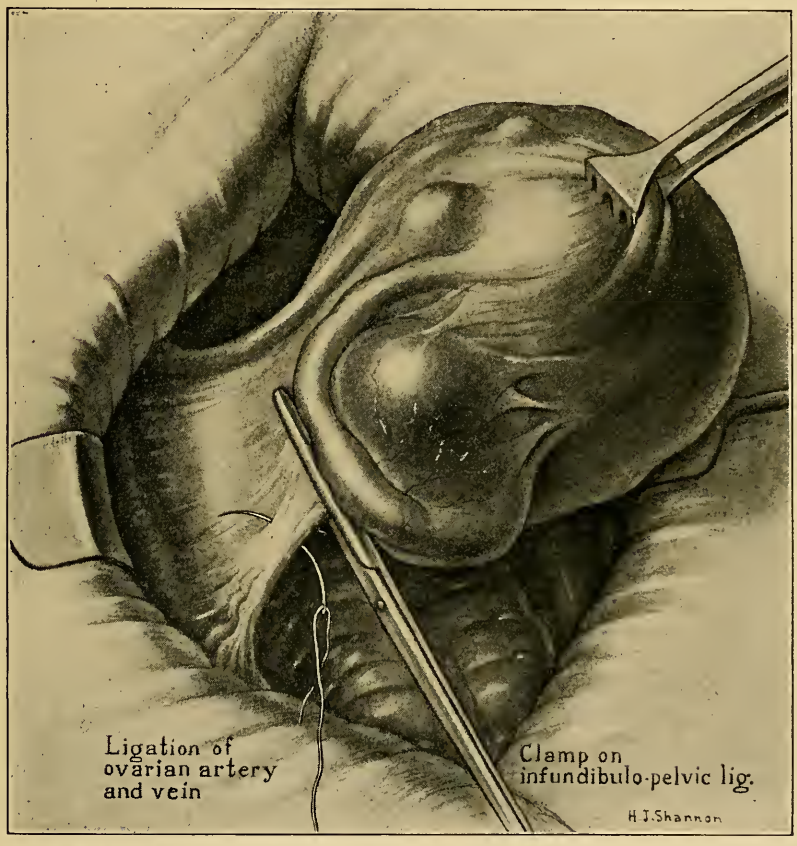

Fig. 69.—The Uterus is Drawn Out of the Wound by a Large Jacobs Forceps.

the uterine incision extraperitoneal. These patients have made smooth recoveries and have menstruated regularly without pain, bladder disturbance, or other pelvic symptoms.

Radical Treatment.—We have seen, in the study of the pathology of chronic tubal inflammation, that the uterus and ovaries are always more or less involved in the inflammatory process, and at times are actually infected and remain infected, even after the source of the uterine and peritoneal infection has been removed. Hence, we may have ovaries, not only in bad company, but actually diseased.

When the ovaries are healthy and the tubes are removed, the circulation in the ovary is more or less impaired, and, unless great care is taken to preserve this blood supply, the ovary, which is retained for its supposed internal secretion, becomes cystic. If, on the other hand, the ovary is diseased, conservation after removal of the tube produces an ovarian pathology that frequently invalids the patient.

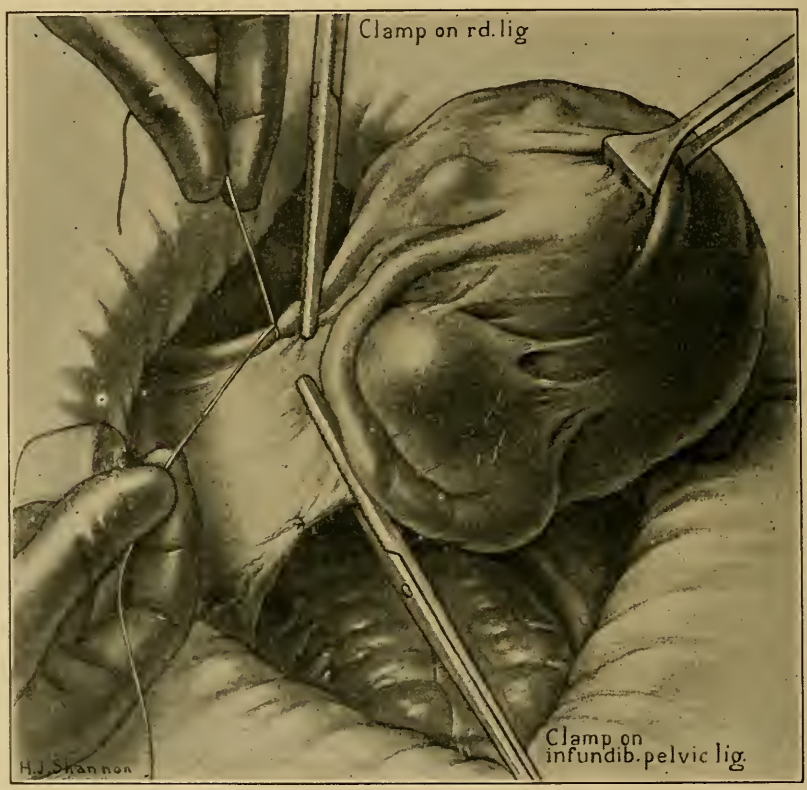

FIG. 70.—A CLAMP IS PLACED ON THE INFUNDIBULOPELVIC AND ROUND LIGAMENTS AND THE LIGAMENTS TIED DISTAL TO THE CLAMP.

The uterus, also, becomes enlarged from continued infection. This enlargement produces a menorrhagia or metrorrhagia, which symptoms will persist as long as the infected organ is retained. Hence, it will be seen that frequently the best interests of the patient are conserved by the complete removal of her pelvic organs. This radical extirpation removes those foci of infection which are beyond nature's reparative powers, and so relieves the woman of her constantly recurring pelvic inflammation.

When hysterectomy is done for chronic pelvic inflammation, it should include the removal of the cervix, for the retention of an infected cervix

acts as a focus for the continuance of parametrial inflammation; besides this, the cervix, with its infected glandular structures, becomes a constant source of leukorrhea. This cervical discharge is just as intractable in the cervix, retained after hysterectomy, as in the cervicitis considered in a previous chapter. The leukorrhea remains a constant nuisance to the patient. Finally, we cannot close our eyes to the possibility of malignant degeneration occurring in the retained cervix.

Technic of the Radical Operation. — When the bladder has been

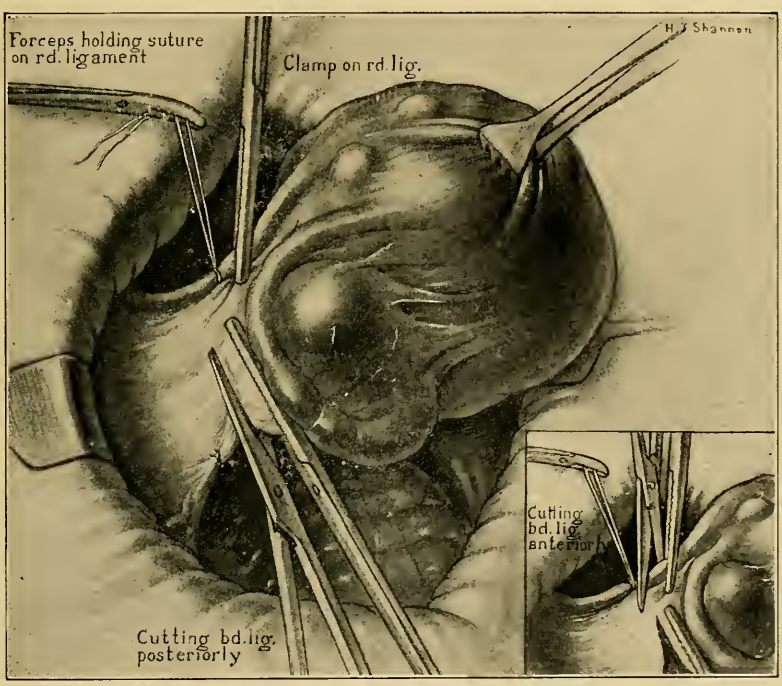

Fig. 71.—The Ligaments are Cut Between the Clamp and Ligature.

emptied, the vagina is rendered sterile by painting the vaginal walls and cervix with tincture of iodin, and the vagina is then packed with iodoform gauze. The patient is now placed in a Trendelenburg posture and anesthetized, the anesthetic being continued while the skin preparation of the abdomen is made. There is considerable advantage in having the patient in the high Trendelenburg posture before the abdomen is opened, as suggested by Guthrie, for, by observing this detail, the intestines tend to gravitate upward from the pelvis, and remain out of the field of operation. When the abdomen is opened, this diminishes the amount of trauma to which the intestine is subjected and consequently the surgical shock.

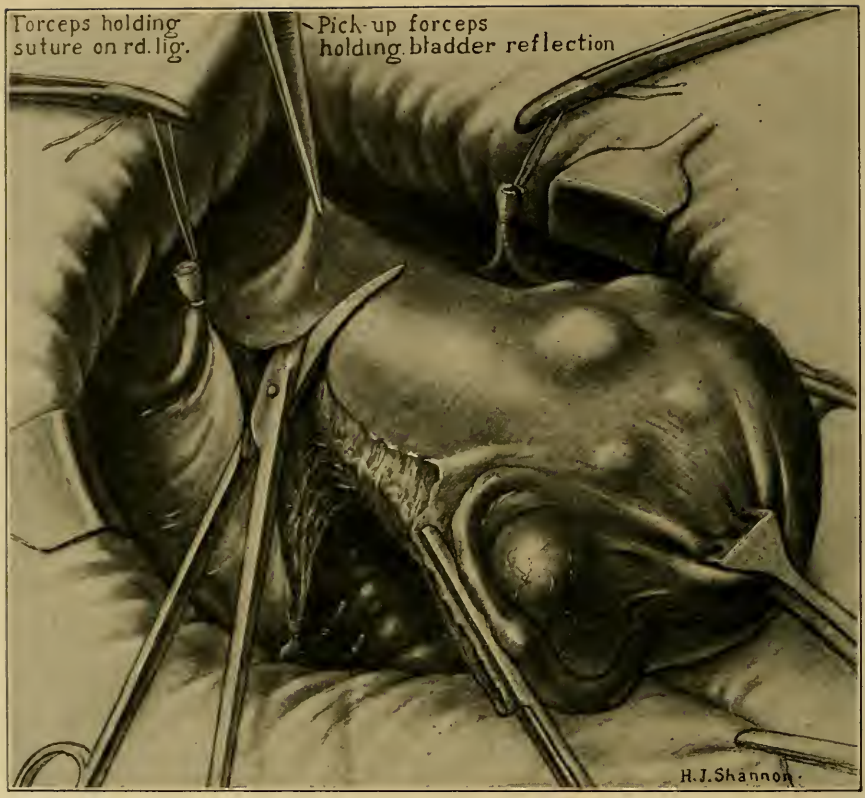

FIG. 72.—A CURVED MAYO SCISSORS IS PASSED UNDER THE VESICAL REFLECTION OF THE PERITONEUM WHICH IS CUT FREE OF THE UTERUS.

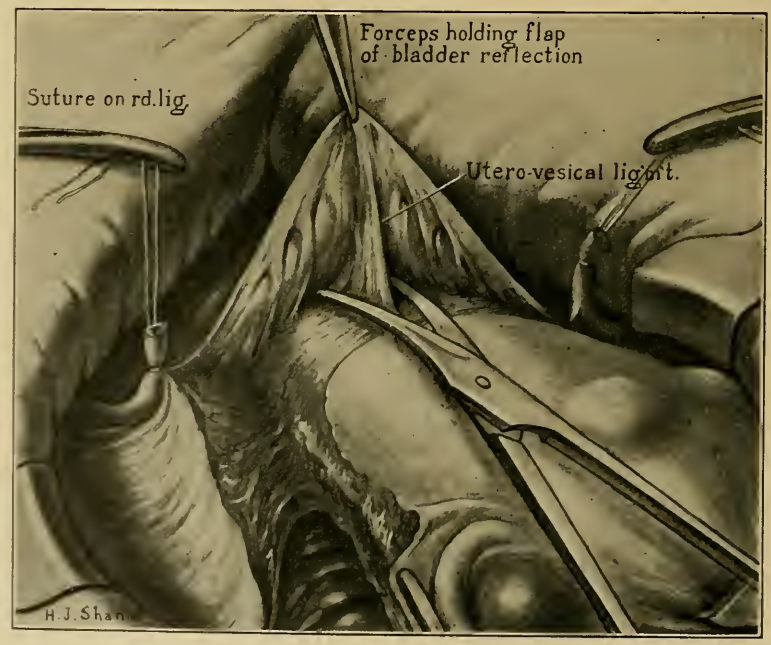

FIG. 73.—ANTERIOR BLADDER REFLECTION CARRIED OVER VAGINAL VAULT.

The abdomen is opened by a long incision in the median line, or just to the right of the median line. The incision should extend from the pubis to above the umbilicus, for *a long incision greatly facilitates the ease of operation.* As soon as the peritoneum is opened, two fingers are inserted in the lower end of the abdominal incision, elevating it. This allows air to enter the peritoneal cavity, and the atmospheric pressure

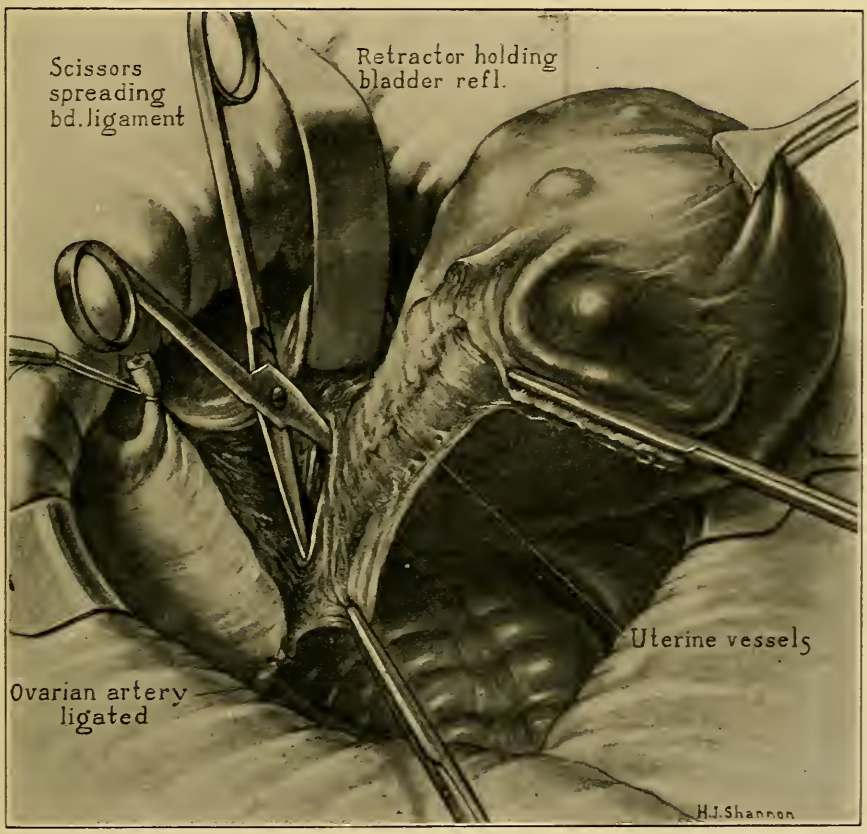

Fig. 74.—The Bladder is Retracted and Uterine Artery Exposed.

causes the intestines to further recede from the field of operation. The wound edges are now protected by towels, and a moist laparotomy sponge is placed across the pelvic brim, so as to protect the intestines from trauma and infection.

The uterine fundus is grasped by a large Jacob's forceps and the uterus is drawn up out of the wound and the adhesions of the tubes and ovaries freed, so that the infundibulopelvic ligaments with their contained blood vessels are exposed. A Kocher clamp is now placed on the infundibulopelvic ligament (Fig. 70), including the ovarian artery and

vein. Distal to this clamp, a suture ligature is passed through the free space in the ligament, and the ovarian artery and vein tied. The ligament is now cut distal to the clamp and the vessels retract. The round ligament is next grasped with a clamp and tied, distal to the clamp, and then severed between clamp and ligature (Fig. 68). This allows the folds of the broad ligament to be separated and the uterine artery and vein exposed and isolated as they come up to the side of the uterus. The

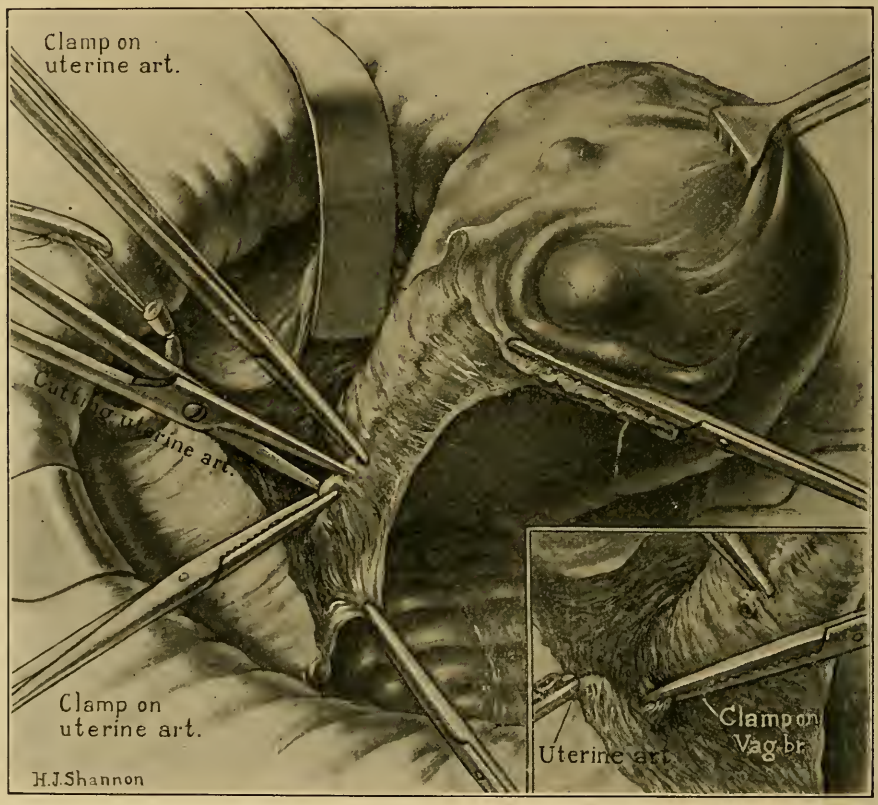

Fig. 75.—Uterine Artery Caught Between Two Clamps and Cut.

same procedure is repeated on the opposite side of the pelvis. We next grasp the round ligament with a pair of pick-up forceps, and with a pair of Mayo scissors, curved on the flat, separate the bladder reflexion of the peritoneum from the front of the uterus (Fig. 72). If the proper line of cleavage is found, which is very superficial, the peritoneum and bladder may be separated without causing any hemorrhage. This is best done by pushing the closed scissors along the line of cleavage and spreading them by opening the blades. In the median line the bladder is firmly attached to the uterus. This attachment must be severed with

the scissors, as illustrated in the figure. After this ligament (Goffe's ligament) is cut, the bladder is readily pushed off of the front of the uterus, vagina, and from the loose cellular tissues at the base of the broad ligaments. With an anterior retractor holding the bladder for-

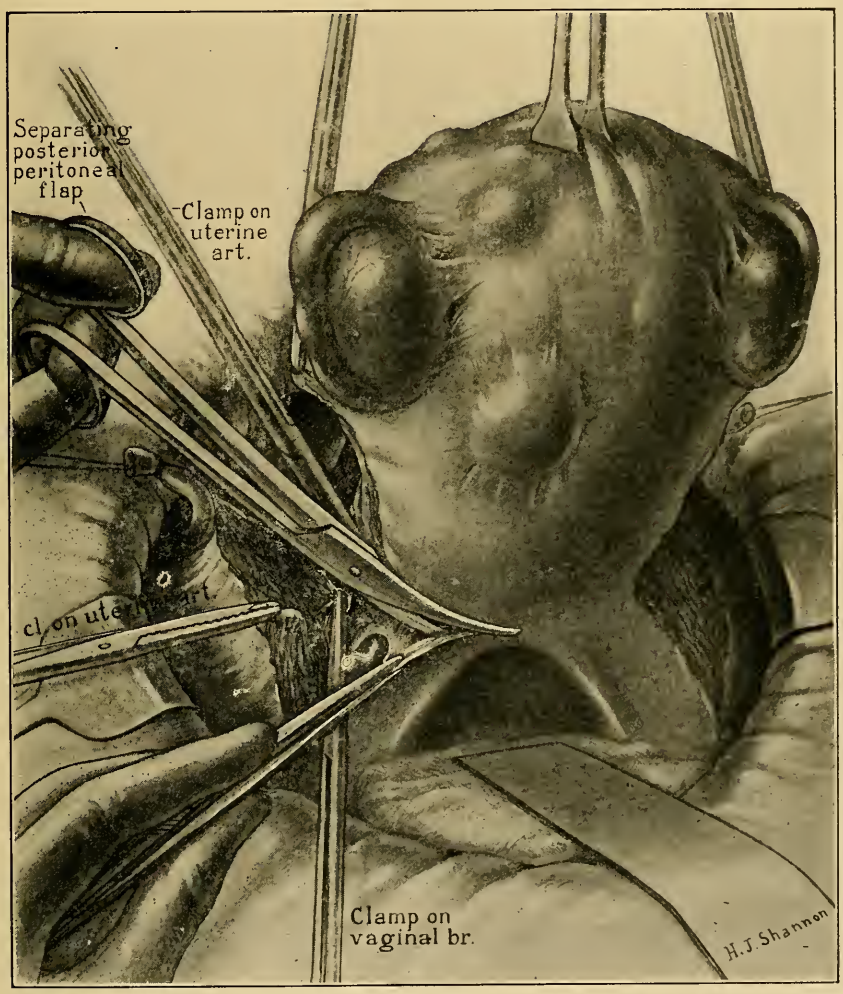

Fig. 76.—The Uterus is Drawn Forward and an Incision is Carried Across the Posterior Peritoneal Fold.

ward, and the uterus well drawn up toward the opposite side, the uterine artery may be easily isolated by blunt dissection with the Mayo scissors. The artery is now clamped in two places and cut between the clamps. Having repeated the procedure on the opposite side, the uterus is drawn forward over the pubis and the attachments of the uterosacral ligaments exposed. Just above the point of their attachment the posterior

sheet of the peritoneum is severed. This allows the uterus to be drawn upward, and the ureters will naturally fall away from the immediate field of operation. With the bladder separated from the front of the vagina and held out of harm's way with a Döderlein retractor, a pair of sharp pointed scissors, curved on the flat, are placed against the anterior vaginal wall, just below the cervicovaginal junction, and forced through

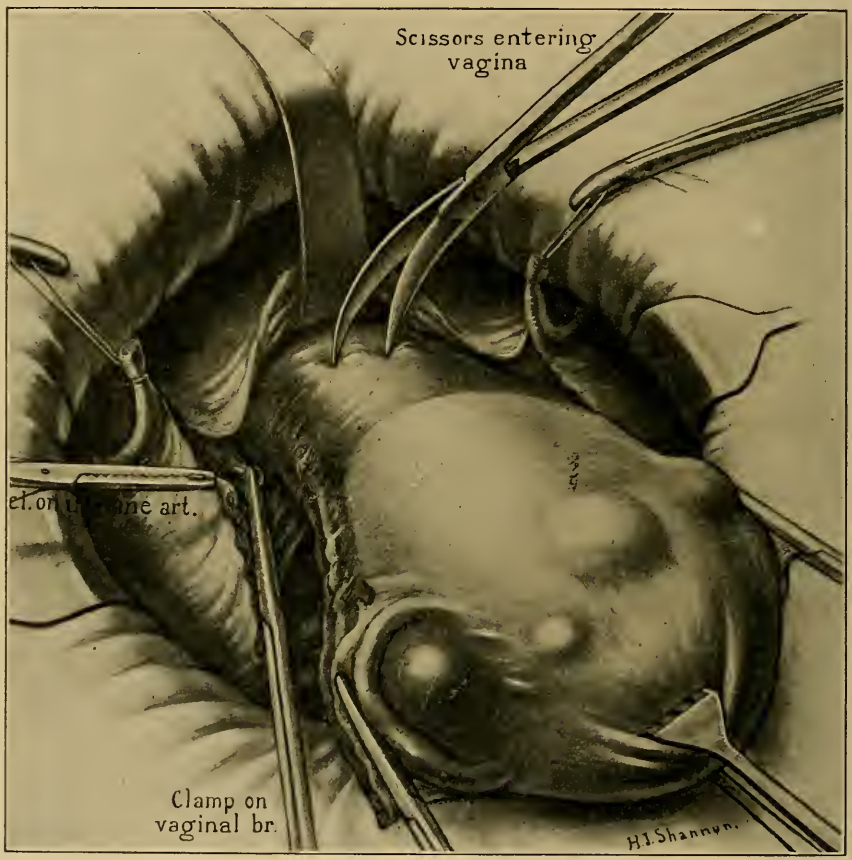

FIG. 77.—CURVED SCISSORS FORCED INTO THE VAGINA.

into the vagina. When the vagina is entered, a Jacob's forceps grasps all coats of the anterior vaginal wall; while the scissors enlarge the opening in the vagina, a second Jacob's forceps seizes the anterior lip of the cervix and draws it up through the vaginal wound. The cervix and upper end of the vagina are now disinfected with tincture of iodin, and a Jacob's forceps placed on the posterior lip of the cervix (Fig. 78). With the cervicovaginal junction now in plain view, it is an easy matter to cut the uterus free at this point with the scissors; as the cervix is sev-

ered from the vagina, a Jacob's forceps grasps the posterior vaginal wall at about the midline.

After the uterus and its adnexa have been removed, the vagina, which is drawn up by traction on the Jacob's forceps, is closed and the cut edges approximated by two figure of eight sutures (Fig. 82). When these sutures are tied, the ends are left long and they act as traction sutures. We now proceed to tie the uterine arteries and the vaginal

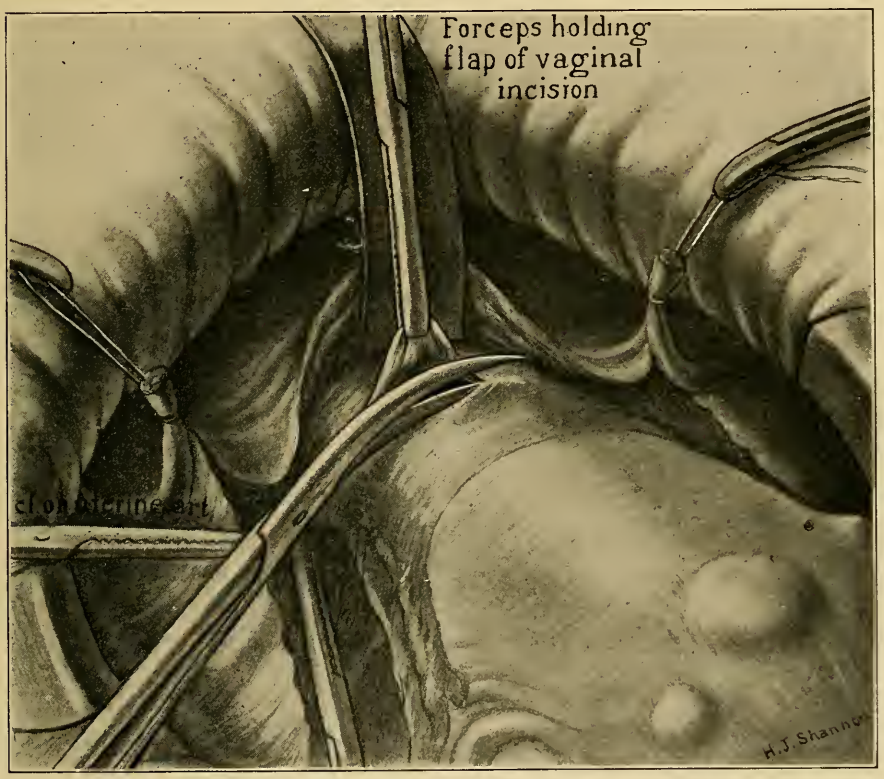

FIG. 78.—SCISSORS ENLARGING THE VAGINAL INCISION AT THE CERVICOVAGINAL SECTION.

branches of the uterine, which are in the grasp of the hemostats. This completes the hemostasis, and, if the preceding detail is observed, leaves the surgeon with a perfectly dry field to peritonealize. A suture is next passed through the muscular coat of the posterior vaginal wall, through the round ligament just to the outer side of the point at which it is ligated, and then passed through the muscular coat of the anterior vaginal wall. When this is drawn taut, the round ligament is sewn into the vaginal vault; the ligament of the other side is treated in a similar manner. We are convinced of the necessity of this detail, for too often

hysterectomy is followed by prolapse of the vaginal walls, unless this point of the technic is observed.

After the round ligaments are securely sutured to the vaginal vault and we are sure that the hemostasis is complete, we are ready to begin the peritonealization. This is done by suturing the posterior fold of the

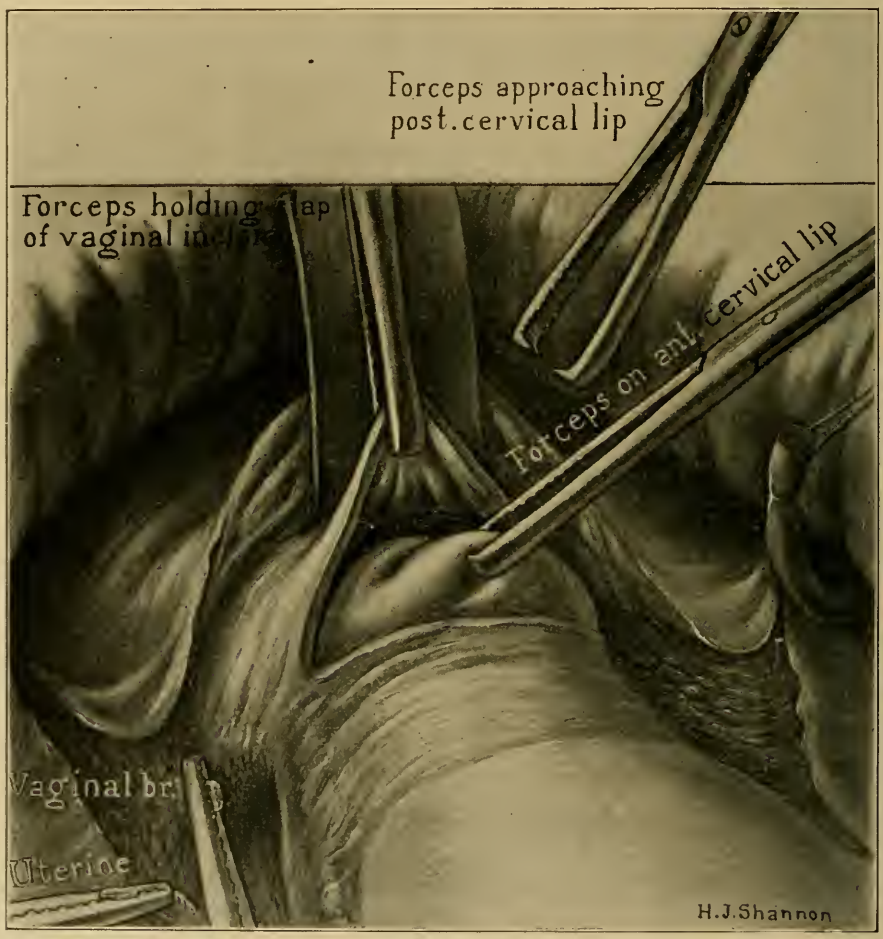

Fig. 79.—The Cervix is then Drawn up through the Vaginal Incision.

peritoneum to the anterior, burying the stump of the ovarian artery (Fig. 85), using a running suture of fine catgut. When the uterosacral ligaments are reached, they are attached to the muscular coat of the posterior vaginal wall, and the anterior bladder reflection is carried over the vaginal vault with the attached round ligaments and sutured to the uterosacrals. This gives the stump an additional support and helps to maintain it high in the pelvis. When the peritonealization is complete,

FIG. 80.—STERILIZING THE VAGINAL PORTION OF THE CERVIX.

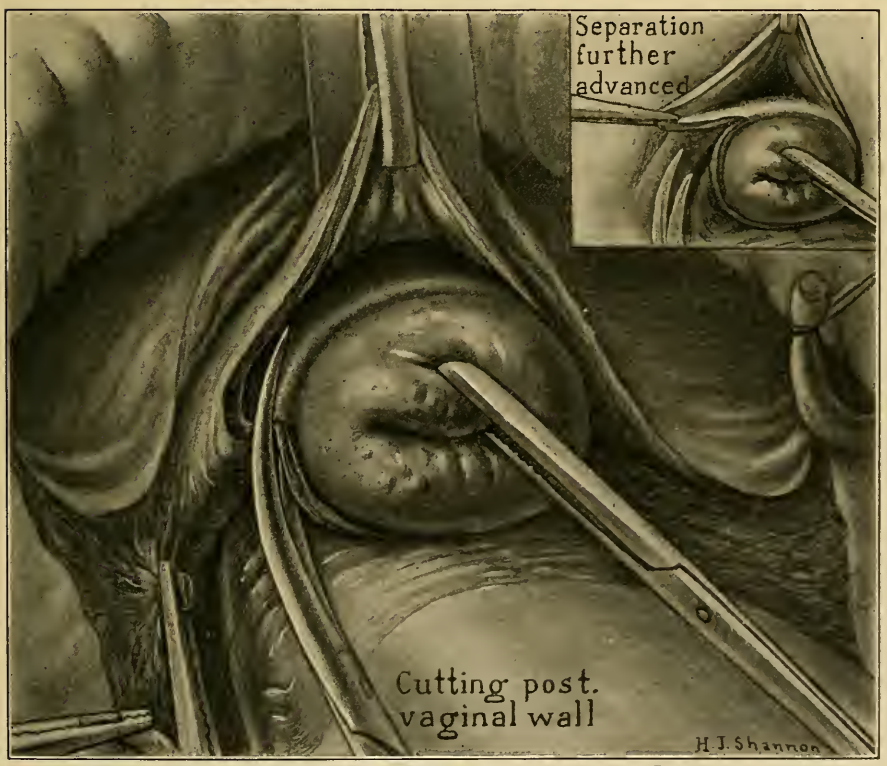

FIG. 81.—EXPOSING THE POSTERIOR CERVICOVAGINAL JUNCTION.

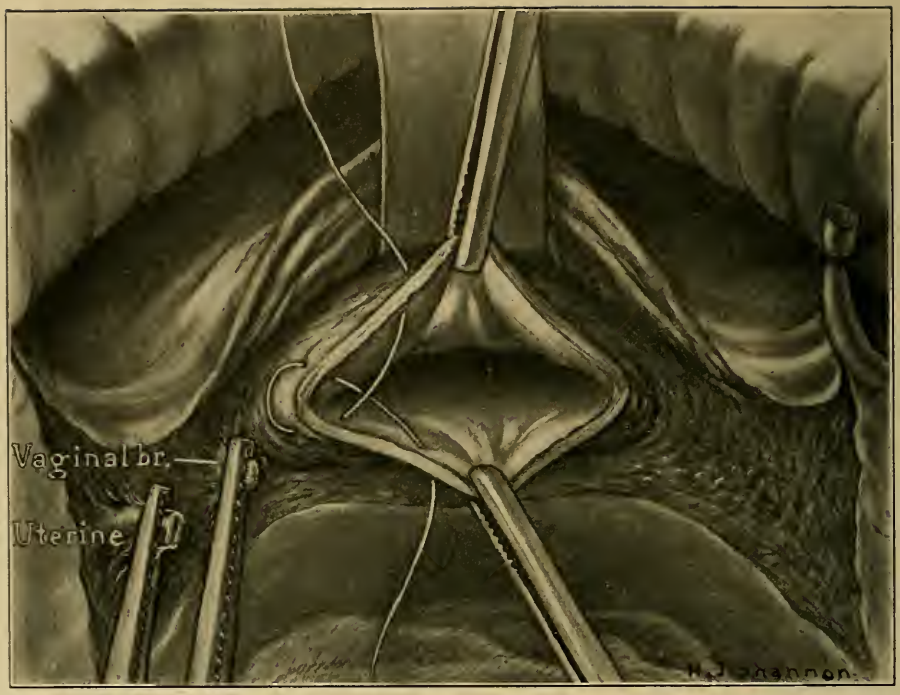

Fig. 82.—Closing the Vaginal Vault with Figure of Eight Sutures.

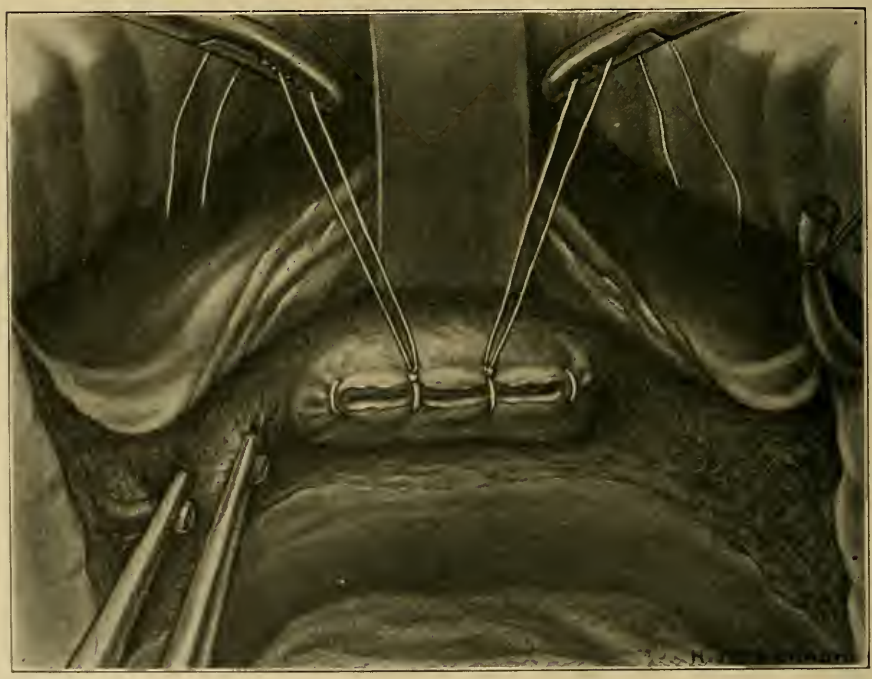

Fig. 83.—Sutures Tied and Used as Tractors.

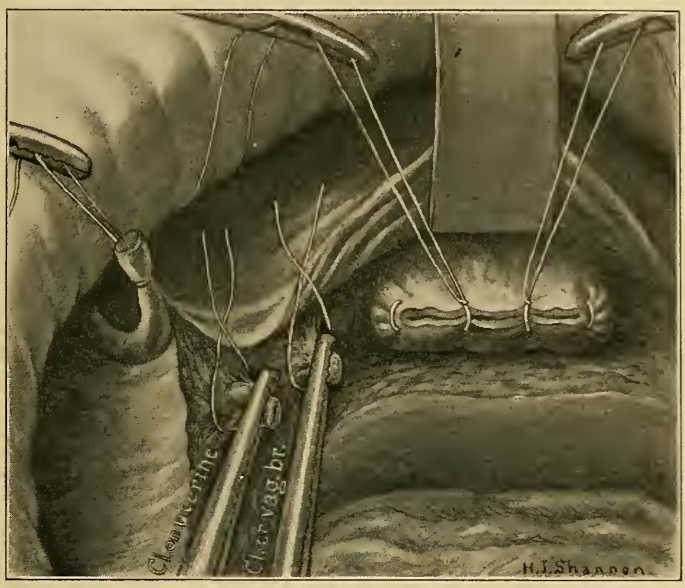

FIG. 84.—LIGATURES PASSED ABOUT THE UTERINE AND VAGINAL BRANCH.

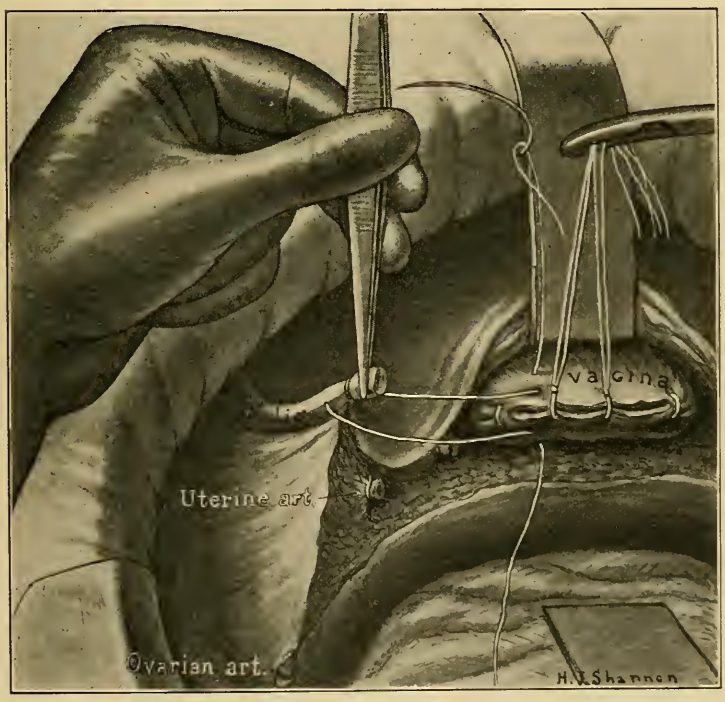

FIG. 85.—DRAWING THE ROUND LIGAMENT INTO THE STUMP.

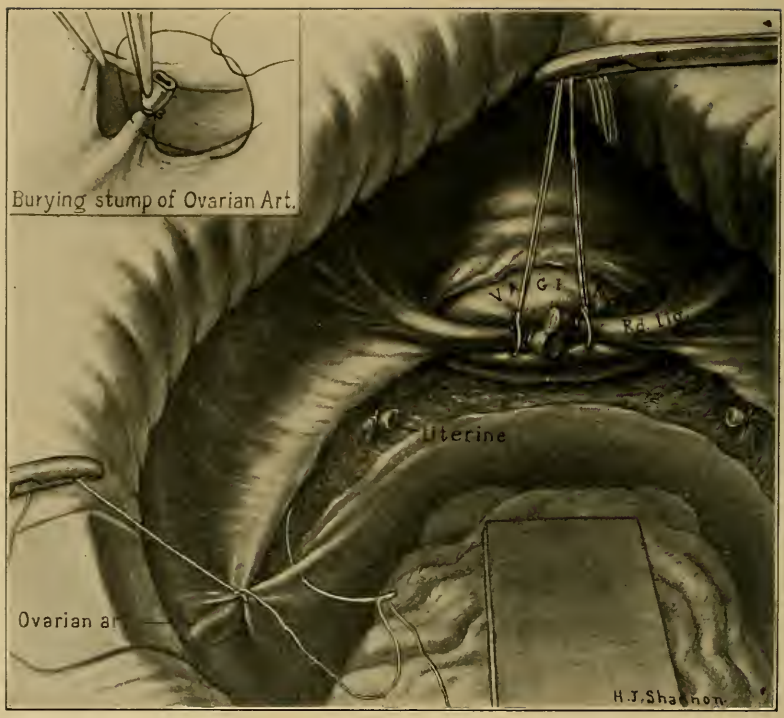

FIG. 86.—ROUND LIGAMENTS SEWN INTO THE VAGINAL STUMP AND PERITONEALIZATION BEGUN.

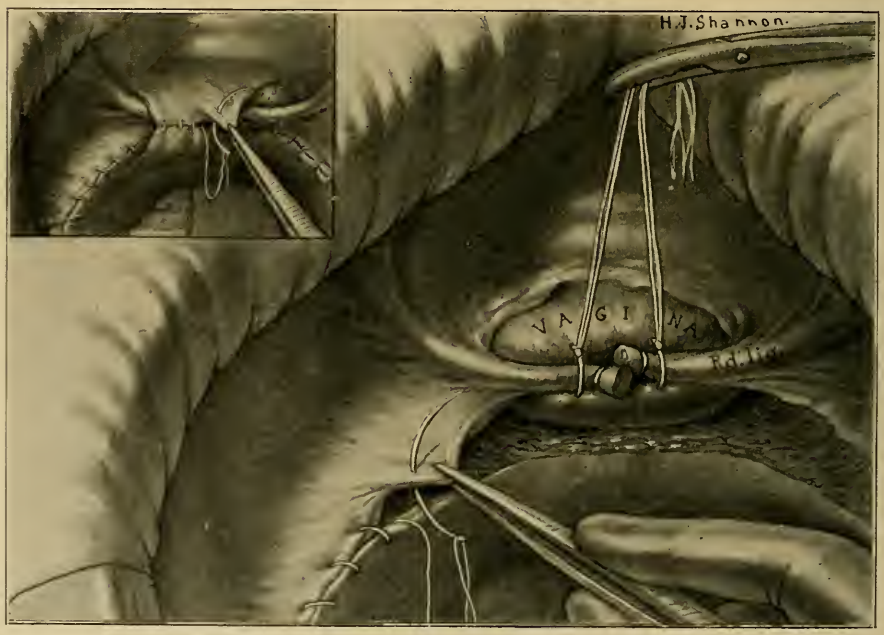

FIG. 87.—PERITONEALIZING THE RAW SURFACES.

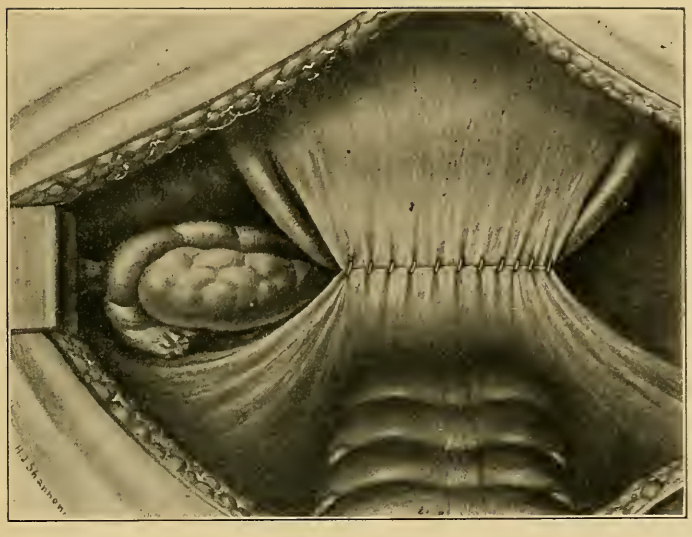

Fig. 88.—Finished Operation When Ovary and Tube Can be Conserved.

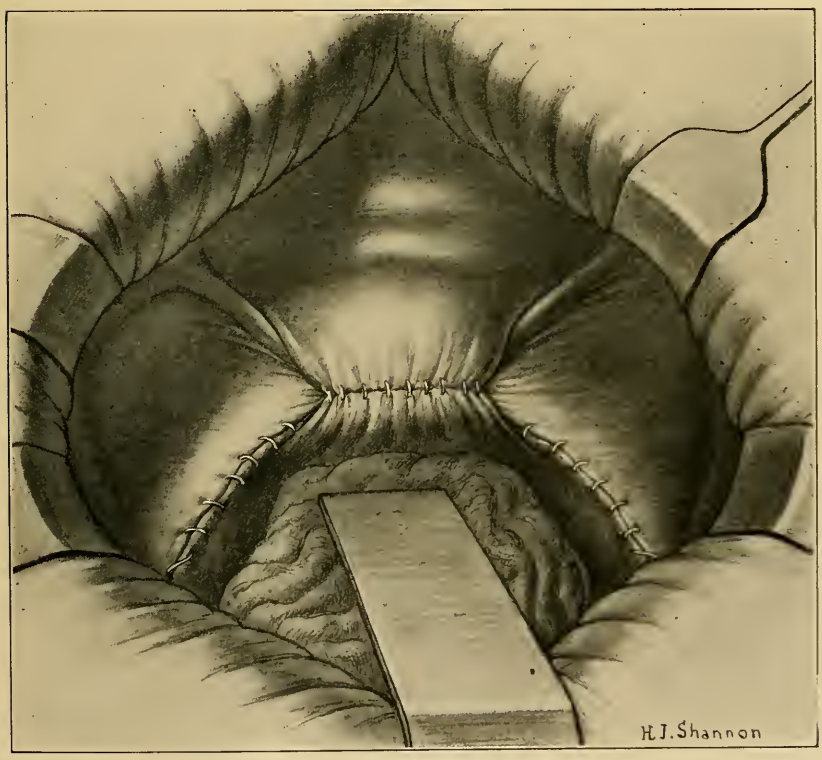

Fig. 89.—The Completed Operation, when Both Ovaries Have Been Removed.

the pelvis should present no raw surface at any point, the suture holes being the only possible point for adhesion.

To our mind the points which give the best success to hysterectomy are (1) the complete removal of the cervix; (2) complete hemostasis; (3) suture of the round and uterosacral ligaments in the vaginal vault; (4) perfect and careful peritonealization of all raw surfaces in the pelvis.

CHAPTER VIII

OVARITIS

Mode of invasion and etiology in ovaritis—Pathology—Usually a history of gonorrheal infection—Chronic ovaritis—Atrophic, hyperplastic, and cystic forms—Sterility—Treatment of chronic ovaritis.

In order to appreciate the pathology resulting from the inflammatory changes which take place in the ovary, it may be well to briefly review the histologic structure of this organ. The ovary is composed in great part of connective tissue, which is continuous through the sheaths of the vessels that enter at the hilum with the connective tissue between the layers of the broad ligament. This continuity of tissue allows an avenue of entrance, through the lymphatics in the broad ligament, for infection from the cervix. Around the periphery of the organ the connective tissue is condensed to form a dense fibrous tunic called the tunica albuginea, which, when unbroken, is protective. On the surface of this tunic is a single layer of low cuboidal epithelium, which is the remains of the original germinal epithelium from which the follicles develop. Underneath the tunica, in the cortical zone of the ovary, lie the ova, each enclosed in its graafian follicle. The blood vessels enter the hilum.

Oöphoritis or ovaritis is an inflammation of the tissues of the ovary. It may appear in either an acute or chronic form; each presents a distinct pathology with terminal results.

Mode of Invasion and Etiology.—Acute ovaritis is produced by an extension of septic or gonorrheal infection from the uterine tube, or an extension of infection from the broad ligament through the lymph channels entering the hilum. Septic ovaritis is most frequently met in the puerperium as a direct invasion of the ovary by infection, after labor or abortion. But bacteria may enter through the hilum from cellular or lymphatic infections in the broad ligament, while in the gonorrheal form, extension is from the tube and commonly produces a perioöphoritis. Occasionally it gains entrance to the interior of the organ by infecting the follicles, and causes an ovarian abscess. Abscess formation, however, is more frequently met with in septic than in gonococcic ovaritis.

Pathogenic bacteria may also reach the ovary, by the hematogenous route, from infective foci in remote regions of the body. This is illustrated in the ovaritis complicating the exanthemata and the ovaritis produced by streptococcic infection of the tonsils (Rosenau). Most of the pyogenic, as well as a goodly number of pathogenic bacteria, have been demonstrated in the ovary. Tuberculous ovaritis is not infrequent, though it is seldom primary in origin, the involvement usually being secondary to tuberculous salpingitis or peritonitis.

Pathology.—In acute ovaritis the ovary is uniformly swollen, reddened, and edematous; it is sometimes enlarged to several times its normal size; the stroma becomes infiltrated with a serous exudate, and small round cells are deposited between the follicles; the follicular epithelium degenerates, the ovum dies and is absorbed, and the liquor folliculi becomes turbid. These exudative processes increase the intra-ovarian tension and explain the exquisite pain and sensitiveness in the ovary which are characteristic of acute ovaritis. At the same time a lymphatic exudate is poured out on its surface which forms plastic adhesions with the adjacent structures. The termination of acute ovarian inflammation must depend on the virulence of the infecting organism and the resistance of the individual tissues. A resolution will follow, if the exudate becomes absorbed, leaving the ovary in an apparently normal condition; on the other hand, the activity of the inflammation may subside, and atrophy of the connective tissue of the stroma take place, with a resulting sclerosis; or again, suppuration may follow, leading to abscesses in the corpus luteum follicles or interstitial spaces, with partial or total destruction of the ovary. Finally, the acute inflammatory process may subside and become subacute or chronic, with a resulting chronically contracted painful ovary, or cystic degeneration of the follicular elements may take place.

Symptoms and Signs.—*There is usually a history of septic or gonorrheal infection of the pelvic organs or of streptococcic tonsilitis or of the exanthemata.* The patient complains of chills or having chilly sensations, of intense agonizing pain and tenderness in the ovarian region (or in both ovarian regions), radiating to the back, down the thigh, and to the breast. There is frequently uterine bleeding (metrorrhagia), occasionally nausea and abdominal distention. If the attack is near a menstrual period there is severe dysmenorrhea, and bowel movements are attended with sickening pelvic pain. Coitus is always painful. Abdominal palpation will elicit tenderness and abdominal tension over the ovarian region, while vaginal exploration, which is painful, shows the ovary to be enlarged, exquisitely tender, and usually prolapsed.

There is nothing else in the pelvis so exquisitely painful as an inflamed ovary.

Except when the infection is a complication of the exanthemata or of tonsillitis, ovaritis does not occur as a separate entity, but occurs as a part of a general pelvic inflammation where the cellular and peritoneal tissues are involved; and the symptoms and signs are those of acute infection, pelvic peritonitis, or tubo-ovarian inflammation; hence the *physical signs mentioned above are not available for making the specific diagnosis of acute ovaritis, except in isolated cases.*

Treatment.—As has already been stated, except when the infection is a complication of the exanthemata or of tonsillitis, ovaritis does not occur as a separate entity, but occurs as a part of a general pelvic inflammation, where the cellular and peritoneal tissues of the pelvis are coincidently involved; hence, *the diagnosis is always complicated by the symptoms and signs of these acute infections.* It will be seen therefore that *the treatment in the acute stage must necessarily be similar to that of acute pelvic peritonitis or salpingitis;* namely, local rest and the relief of the intense ovarian pain, which is produced by the increased intra-ovarian tension, consequent upon the edema and cell proliferation which occurs within the tense unyielding ovarian capsule. The Fowler position, with the application of heat or cold applied directly over the ovarian region, and the administration of morphin, will relieve the intense pain and tend to localize the inflammatory reaction. As the ovary, when it is the seat of an infective inflammation, always tends to drop into the *cul de sac,* bowel movements are intensely painful, because of the close proximity of the swollen painful ovary; hence, the use of cathartics should be avoided and the lower bowel emptied with small non-irritating enemata. In our experience, active counterirritation, either over the ovarian region or applied to the vaginal vault, has not given any relief of the pain. *Not until the acute symptoms* produced by the perioöphoritis and the associated peritonitis *have subsided, should any local applications be made to the vaginal vault.* It will be seen, therefore, that aside from the relief of pain, *time alone is the factor which contributes to the resolution of the acute ovarian inflammation.*

After the acute symptoms are quiescent, hot vaginal douches of 120° F. will hasten the absorption of the peri-ovarian exudate. If an abscess forms, it will be shown by the persistence of an evening temperature, increased size of ovary, and a leukocytosis. Drainage should be made by vaginal section; this may be followed later by oöphorectomy, when all acute symptoms have subsided.

Chronic Ovaritis.—Chronic ovaritis may be the terminal stage of

an acute infection, or may result from any condition which produces and maintains a long continued congestion of the ovary, as, for example, retrodisplacements of the uterus, subinvolution, constipation, masturbation, sexual excesses, etc. All of these conditions interfere, more or less, with the efferent circulation and produce a chronic venous stasis, which ultimately results in either

(1) Atrophic oöphoritis,
(2) Hyperplastic oöphoritis, or
(3) Cystic oöphoritis.

In the *atrophic form,* which is inflammatory in origin, the principal structure involved is the stroma, but often the parenchyma also participates in the local tissue changes. There is an overgrowth of connective tissue and the follicles atrophy from pressure. This form is recognized by the following characteristic gross appearances: the ovary is of small size, sclerotic, having a nodular surface and a thick tunic; there are practically no follicles.

The *hyperplastic form* of chronic ovaritis is a result of stasis, and hyperplasia of the stroma results. It is recognized by its increased size and the nearly smooth white surface. On section we find the tunic immensely thickened and the stroma dense, with a few follicles in the deeper structures.

The *cystic ovary* is the direct result of passive congestion and hyperplasia of the stroma; the ovary is large and presents irregular groups of cysts throughout the gland, which give the ovary an irregular shape. These follicular degenerations have been attributed by Rosenau and Davis to early hematogenous streptococcic infection. Clinically, it would seem that the thick tunic and dense stroma prevented the natural maturation of the follicle, consequently numberless immature follicles are arrested in their progress toward the surface. The thickened tunica albuginea and the degeneration of the follicles explain the sterility.

Symptoms.—There is usually pain in one or both ovarian regions, which is aggravated by standing or walking and before and during menstruation. The pain sometimes extends to the back and down the thigh and *there is dyschezia, especially if the left ovary is prolapsed* and the sigmoid and rectum are loaded with fecal masses. Dysmenorrhea is a constant symptom of chronic ovarian disease, and pain precedes the appearance of the flow and the ovarian soreness usually continues after the flow has ceased. Marital relations are always painful and even the introduction of the douche nozzle causes pain. With large cystic ovaries there is not infrequently some anomaly in the menstruation. This may

take the form of a menorrhagia or what is more usual a metrorrhagia, with a more or less continuous spotting after the normal flow has ceased. In atrophic ovaritis there is amenorrhea. Premenstrual pain and enlargement of the breast, hysteria and, hystero-epilepsy are often associated with chronic ovarian inflammation. *Sterility is frequent,* owing to the destruction of the follicular elements or the thickening of the fibrous capsule, which prevents normal maturation of the ovaries.

Physical Signs.—Pressure at Morris' point usually elicits pain, and on bimanual examination the ovary is felt as a large, tender mass, low down in the lateral or posterior fornix, usually more or less movable, distinct and not blending with the uterus or parametrial tissues; or the ovary may, as in the atrophic form, be smaller than normal, adherent and very sensitive.

A large *ovary may be normal, its increased size being due to a simple transitory corpus luteum cyst; while a small ovary may be pathologic.*

Recto-abdominal examination is of great value, for by the rectal touch, one is often able to palpate the ovary with great facility.

Prognosis.—The prognosis in chronic ovaritis should always be guarded, for *the ovary,* like the tube, has *great reparative powers.* A spontaneous symptomatic cure may occur at the menopause, though seldom before, unless absolute sexual abstinence is observed. Abscess formation may result, usually after puerperal infection, or the ovary may be changed into a mass of small cysts, with little or no stroma remaining. Sterility is a common sequel of chronic ovaritis. The pathologic ovary never completely regenerates.

Treatment of Chronic Ovaritis.—The treatment consists of rest in the recumbent posture, especially at the menstrual period, and counterirritation. This may be made with a capsicum plaster or the application of iodin to the abdomen over the ovarian region and to the vaginal vault. The local treatment to the vaginal fornices by iodin and the introduction of elastic wool tampons saturated with boroglycerite, may be repeated once in three days. These treatments are best given with the patient in the knee chest position, for in this way the ovary can be raised by the tampon and its circulation improved. The daily employment of hot douches and the keeping of the lower bowel empty tends to improve the pelvic circulation. The bowels should be emptied by small doses of cascara sagrada and small saline enemata. Hot sitz baths and mud baths have been employed in the foreign clinics for years, frequently with some degree of success.

For the pain, certain antispasmodics, as the chlorid of gold and sodium gr. 1-10, or extract of cannabis indica in ¼ gr. pills, or the tinct.

of pulsatilla ♏ x, may be given three or four times a day, beginning a week before the time of the expected period, and continued during the flow. Latterly we have used aspirin gr. v, with ext. ovarii gr. ii, in the same manner, with apparently greater relief. The importance of the restoration of the general health by hygiene, air, and tonics should never be forgotten in the management of these patients.

When local and systemic measures fail, surgical intervention may be employed; this includes liberation of the peri-ovarian adhesions, linear incision through the fibrous capsule, ignipuncture, wedge shaped resection of the diseased structure, and, in extensive structural change, oöphorectomy.

We have long since come to the conclusion that ignipuncture and resection are not curative procedures and frequently leave the ovary badly scarred, adherent and painful, subsequently necessitating its removal. *Probably no condition in woman places a greater responsibility on the surgeon than the determination of what disposition he shall make of an ovary in a given case.*

A cystic ovary is not always a diseased ovary, for cysts when single, few, superficial, and rounded, are usually corpus lutein cysts or dropsical follicles and do not require surgical treatment. When the cysts are crowded together in masses of irregular shape, and, on splitting the ovary to its hilum, are found throughout the gland, the ovary should be extirpated.

Small hard atrophic ovaries giving constant premenstrual and intermenstrual pain demand oöphorectomy; however, a pure neurosis is not an indication for ablation.

Hypertrophy of the ovary may be the result of a chronic inflammation, but is more often due to a chronic hyperemia of mechanical origin, such as the large ovaries found in pregnancy or with fibroid tumors. Their stroma is made up of fibrous tissue, rich in spindle shaped cells, with a lack of primordial follicles. Such ovaries do not necessarily, because of their size, demand radical surgery. Suspension of such an ovary will reëstablish its circulation and still retain the ovarian function.

On general principles, resection of the ovary should be discouraged.

CHAPTER IX

PELVIC PERITONITIS

Causes of pelvic peritonitis—Gonococcic pelvic peritonitis—Pyogenic peritonitis—Symptoms; chill, pain, pulse, muscular rigidity, abdominal tension, tympanites, nausea and vomiting, constipation—Physical signs—Treatment of pelvic peritonitis—Arrest of intestinal peristalsis.

No discussion of pelvic infections in women would be complete without a consideration of peritonitis as a pathological entity, for the peritoneum is involved in almost all inflammations of the uterus, parametria, tubes and ovaries as an extension and complication of the original inflammation, and when it occurs, presents a definite pathology and a typical symptomatology.

Broadly speaking, peritonitis is an inflammatory reaction on the part of the peritoneum against trauma and invasion by pathogenic organisms, and when beginning in or confined to the pelvic peritoneum, it is usually the result of an extension of infection from the uterus through the tubes or through the parametrium.

Causes.—Its causes may be septic, gonorrheal, or tuberculous infection. It is always secondary to infections of the uterus, tubes, ovaries, bladder, pelvic cellular tissues, intestines or vermiform appendix. Occasionally it is due to septic infection following abdominal section.

Gonorrheal and tuberculous peritonitis nearly always proceed from infection of the tubes; on the other hand, septic peritonitis may result from an infection through the tubes or an extension through the lymphatics of the uterus and parametrium.

Gonococcic Pelvic Peritonitis.—Gonococcic pelvic peritonitis is primarily the reaction of the peritoneum covering the tubes, and the reaction produced in the peritoneum of adjacent viscera coming in contact with them.

When the gonococcus invades the interior of the tube and finds a suitable environment for its multiplication, it excites an endosalpingitis, the underlying muscles become infected, and then the infection is spread through the lymphatics to the peritoneum, producing a perisalpingitis and pelvic peritonitis. As the infiltration of the tube wall increases, an abundant fibrinous exudate forms, extending into the subperitoneal tis-

sues, and as the tissue reaction continues, there is marked dilatation of the vessels, swelling of the endothelium, and an exudate is poured out on the serous surface; or, fluid bearing gonococci may escape from the free fimbrial end of the tube and *directly infect the contiguous peritoneum and ovarian surface.*

The thickness of the tube wall is of necessity enormously increased by exudate and the infiltration of leukocytes. In the early stage the polynuclears predominate, while later the mononuclears are more abundant. As the retrogression takes place, the polynuclears decrease, while the mononuclear cells relatively increase.

Naturally the amount of exudate which forms on the surface of the peritoneum will vary with the intensity of the peritoneal reaction. If the irritation is not very intense, a thin fibrin deposit results, which develops into thin delicate fibrous tissue strands (cobweb adhesions). In the more virulent inflammation a very large amount of coagulable material is exuded. This is granular for the most part; but on the surface of this there is a layer of fibrinous material, which results in permanent organization. In other words, the granular substratum is absorbed while the fibrinous material on the surface organizes, forming a fibrous membrane, binding tube, ovary, and intestine into one conglomerate mass, the result of the general peritoneal reaction.

Fortunately, in the gonococcic form this reaction is confined almost invariably to the peritoneum of the pelvis and that covering the viscera contained therein, as, owing to the formation of fibrinous adhesions, the pelvic cavity becomes walled off early in the process by adhesion of the omentum, sigmoid, and parietal peritoneum and the *inflammatory reaction becomes localized in the pelvis.*

Pyogenic Peritonitis.—The peritoneal reaction which takes place after labor, abortion, or intra-uterine manipulation or instrumentation, is usually due to the invasion of the streptococcus or the staphylococcus. In this form of infection, the reaction may be confined to the pelvis or *may spread to the general peritoneal cavity.*

Bacteria gain entrance through the genital tract by way of the uterus or through lacerations of the cervix or vagina, and reach the peritoneum through the lymph channels by contiguity without previous focal involvement; or, the peritonitis may be secondary to some lesion where the invading organisms are temporarily halted. For, while the endometrium is the chief port of entry, the infection rapidly spreads through the veins and lymph channels, not only of the endometrium, but also of the parametrium, thus ultimately reaching the peritoneum. The escape of infective material from acutely inflamed tubes may be the source of the

peritoneal involvement; or, a walled off pelvic abscess, or an abscess in the wall of the uterus may leak or rupture, and spread the infection to the adjacent peritoneum.

On reaching the surface of the peritoneum, an inflammatory reaction to this type of bacteria is rapidly diffused over its surface. The extent of the diffusion depends on the virulence of the infecting organism and the resistance of the tissues (the peritoneum).

The surface of the peritoneum is reddened, the endothelium swollen and lusterless and covered with a thin flocculent exudate, or the exudate may be serous, or seropurulent, containing numberless bacteria. Few adhesions are formed, owing to the rapidity of the reaction and the absence of a fibrinous deposit. This explains the tendency of this form of peritonitis to spread upward into the general peritoneal cavity and cause a fatal issue.

Symptoms.—*Chill.* In the acute form the disease may be ushered in with a chill or chilly sensations, intense pelvic pain and tenderness, high pulse and temperature, leukocytosis, tympanites, irritability of the bladder, and dysmenorrhea. A distinct chill or *chilliness is more common in the pyogenic* than in the gonococcic form, for *in the former the blood stream is often invaded* through lymphatic extension. The initial chill may be decisive and single or may be repeated. Preceding or following the chill, there is rise of temperature and a leukocytosis. The fever is relatively low in the gonococcic form, ranging from 101-103° F., usually higher in the puerperal form. The leukocyte count rapidly rises to 20,000 or 25,000, and the polymorphonuclear percentage is 85 or more. In the gonococcic form, leukocytosis is not quite so marked, and the percentage of polymorphonuclears is less.

Pain.—The patient complains of intense pelvic *pain as soon as the peritoneum is involved.* This pain is always characteristic of peritoneal irritation and is *sharp and cutting.* At first it is localized in the region of the focus of origin, and then is diffused over a wider area; this may be due to reflex diffusion of sensation, or to the extension of the peritoneal hyperemia. The pain is always aggravated by motion, by intestinal peristalsis, and by emptying the bladder or rectum, owing to the hyperemic condition of the serosa. *Motion allows the sore surfaces to rub together* and causes pain such as is observed when the pleura is inflamed. In the gonococcic form, the severe pain usually subsides in twenty-four to thirty-six hours, if the intestinal tract is kept at rest. On the other hand, in pyogenic peritonitis there is intense pain beginning in the pelvis which rapidly spreads over the whole abdomen. It is continuous, and is increased by movement or external pressure. *Pain is*

increased by suddenly releasing the pressure of the palpating hand. There is always excessive abdominal sensitiveness, except when the initial intoxication is so overwhelming that the terminal nerves are insensitive to peripheral irritation; then the patient makes no spontaneous complaints of pain.

Pulse.—The pulse is always accelerated. The rate, however, in gonococcic peritonitis is not out of proportion to the rise in temperature, while in streptococcic invasion the pulse not only becomes more rapid, but progressively increases in rate, breaking its relation with the temperature rise, and, because of the absorption of toxins or blood invasion by the offending organisms, the cardiac muscle is weakened and the quality of the pulse is always impaired.

Muscular Rigidity—Abdominal Tension.—Early in the attack, on abdominal examination one may find the entire *abdomen distended and rigidity of the muscles* in the hypogastrium and over both lower quadrants. This is true, even in the absence of diffuse pain. As the disease subsides, the rigidity becomes limited to the lower portions of both recti. On the other hand, if the *inflammatory process tends to spread, the muscular tension increases and extends over a greater area.*

Tympanites.—The amount of abdominal distention is always considerable, but in peritonitis occurring during the puerperium, because of the laxness of the abdominal walls, the distention is extreme. *It is most marked where there is a rapid diffusion of the infection.* As the disease progresses, the *intestinal wall becomes edematous and infiltrated,* due to hyperemia, resulting in non-function of the muscle and nerves, hence the tympany increases. When the activity of the process subsides, the intestine gradually regains its muscular tone and the meteorism progressively decreases.

Nausea and Vomiting.—Nausea and vomiting are neither common nor persistent in pelvic peritonitis, for it may be stated that, the *further the inflammatory reaction is removed from the duodenum and stomach, the less prominent is the symptom of vomiting.* In spreading peritonitis, gastric irritation may become a symptom of great importance.

Constipation.—The bowels are usually inactive in the early stages of pelvic inflammation, owing to the paresis of the terminal portion of the large bowel. This is often due to the amount of fibrinous exudate thrown out in the peritoneal reaction, or, as before said, to the edema and infiltration of the intestinal wall. As the inflammatory changes subside, the bowel gradually regains its muscular tone, and is readily emptied with small enemata.

Physical Signs (Pelvic).—On bimanual examination there is fixity of the pelvic organs. At first, this is due to hyperemia of the peritoneum and muscular spasm, but in thirty-six or forty-eight hours the whole pelvis is filled with a plastic exudate and the whole pelvis becomes exquisitely sensitive.

This exudate fixes the contents of the pelvis as in a plaster cast, and fills the *cul de sac* and the pelvis on either side of the uterus. As the mass becomes more dense, the sense of tumor is more pronounced. In the streptococcic form, pelvic examination gives less evidence of mass, for the exudate is serous or seropurulent, *not plastic.* But motion of the cervix, however, is extremely painful.

The density of an inflammatory tumor is often misleading, owing to its extreme hardness; it may even simulate the density of a carcinoma. The bulk of these inflammatory tumors is not produced by the exudate, either in the tube or around it, but by an extensive edema of the subperitoneal connective tissue. *This fact is often lost sight of and a pelvic abscess diagnosticated and an unnecessary operation advised.*

Time will effect the most miraculous absorption of these tumor masses. When the inflammatory reaction subsides and the edema and infiltration lessen, the *mass becomes hard and insensitive and the leukocyte count falls.* Should an abscess form, the mass generally increases in size, the pain recurs, and the tumor becomes more sensitive. It may bulge into the *cul de sac* and displace the uterus forward and upward. Occasionally it is possible to elicit fluctuation. The evening temperature is always elevated, the patient perspires freely in the remissions, and the leukocyte count is relatively high.

Diagnosis.—Certain symptoms are common to all forms of peritonitis, temperature, tenderness, tension or rigidity, abdominal sensitiveness, tympany, intestinal paresis and gastric irritation, and they differ only in their intensity and location.

Pelvic peritonitis has to be differentiated from extra-uterine pregnancy, appendicitis, and ovarian cyst with a twisted pedicle. (This differentiation has already been referred to in the chapter on salpingitis.)

Prognosis.—The prognosis will depend largely on the type of the infecting organism and the resistance of the individual. In the gonococcic type the peritoneal reaction usually results in resolution, with complete or partial symptomatic and anatomic cure. Unfortunately, owing to an improper understanding of the clinical course of gonorrheal infection of the peritoneum, many cases are overtreated and mixed infections ensue. These lead to the formation of peritoneal and

visceral adhesions of the fibrinous type, with encysted serous effusions, pelvic abscess and spreading or diffuse peritonitis.

The outcome in puerperal peritonitis is always more serious, for during pregnancy, and the puerperium, the lymphatics are more active and the streptococcus and staphylococcus are more virulent organisms. While proper treatment and timely intervention may arrest the advance in many cases of spreading peritonitis, we doubt that a really diffuse case ever recovers.

The difficulty in forming any accurate idea concerning the outcome of reported cases lies in the fact that observers differ so widely in their interpretations; thus, many cases of spreading peritonitis are reported as diffuse. Puerperal cases, furthermore, are always complicated by more or less blood stream infection, and consequently the heart, liver and kidneys are subject to greater degenerative changes and thus diminish the resistance of the individual.

To summarize, we would say that the prognosis in *gonorrheal* pelvic peritonitis, under proper treatment, is *good as to life;* but there is always some remaining pathology, which may or may not produce pelvic and abdominal symptoms. On the other hand, puerperal peritonitis, with its tendency to spread into the upper abdomen, and the associated infections in the uterus, parametria and pelvic veins, always *makes the outcome as to life serious.*

Treatment of Pelvic Peritonitis.—The management of pelvic peritonitis will be considered under the heads of (a) palliative and (b) operative.

In pelvic peritonitis, the type of the infection not only helps to determine the prognosis, but suggests the plan of treatment to be adopted. In the gonorrheal type, with its typical history and clinical course, the tendency is toward a localization of the process and a spontaneous and symptomatic cure. This, of course, may be followed by acute exacerbations of the old process; on the other hand, *in pelvic peritonitis of puerperal and postabortal origin, the inflammatory reaction has a greater tendency to spread* and involve a greater area of the peritoneum, and consequently is more apt to demand surgical measures to aid the arrest of the inflammation.

It has been difficult until recently to make the surgeon understand that there is a difference between the peritonitis following perforation of the appendix, or a gastric or duodenal ulcer, and the peritoneal reaction which takes place as an extension of a tubal inflammation or as a complication of a parametric process. *In the former, the intestinal content is suddenly poured into the unprepared peritoneal sac* and the

material is diffused over a large area; consequently the reaction on the part of the peritoneum is insufficient to localize the process. In *pelvic peritonitis,* except in the presence of a very virulent infection, the *reaction is but an extension of the tubal process,* beginning as a perisalpingitis, and the adjacent peritoneum has already become hyperemic and has poured out an exudate which tends to cause adhesions of the contiguous viscera and thus localize the process within the pelvis.

In the former, surgical intervention may remove the cause and thus stop the supply of infective bacteria and toxins, and, by properly employed drainage, favor the localization of the peritoneal reaction and thus change a spreading peritonitis into a localized peritonitis. On the other hand, in pelvic peritonitis, localization of the reaction within the pelvis has already taken place, the pelvis being cut off from the general cavity by the adhesion of the sigmoid to the uterus and the bladder, and, in order to remove or drain the focus, the surgeon is compelled to spread the infection throughout the lower abdomen; hence, palliative measures have their greatest field in the pelvic type of the disease.

It is the desire of every surgeon that the inflammatory reaction of the peritoneum be localized. This may be greatly assisted, except in such abdominal calamities as perforations of the hollow viscera, by

(a) Rest,
(b) Posture,
(c) Arrest of intestinal peristalsis,
(d) Opium for the relief of pain,
(e) Enteroclysis.

Rest in the semirecumbent posture is the most comfortable position for the patient, for the flexion of the thighs relieves the strain on the tense abdominal muscles. When we add to this the Fowler elevated trunk posture, as may be obtained with the Gatch bed, we not only favor gravity drainage, by making the pelvic peritoneum the lowermost point in the peritoneal sac, but actually tend to localize the peritoneal reaction in the true pelvis. This is done by *adhesion of the omentum to the bladder and anterior parietal peritoneum, and attachment of the loop of the sigmoid to the fundus and the bladder reflection.* Thus, any low typed infective reaction within the pelvis, or the involvement of any of the viscera contained therein, is actually limited to the structures below the brim.

Unfortunately, in peritoneal reactions of the puerperal type, owing to the arrest of involution, *the uterus* is of larger size and *is usually well out of the true pelvis.* This, together with the greater virulence of the

infective organisms and the increased size of the lymphatic channels, consequent upon the pregnant state, offer larger channels for absorption; therefore, the infection is more difficult to confine within the pelvis and we are more likely to meet with the spreading type, for the uterus with its perimetritis is carried into the field of diffusion. Even in this type, clinical experience has convinced us that there are advantages from the routine use of the Fowler posture.

Intestinal peristalsis must be arrested, for motion of the intestines rubs the hyperemic surfaces together, and always causes or increases the peritoneal pain; besides this, *the peristalsis diffuses the infective material,* and thus causes an extension of the inflammatory reaction in the peritoneum.

To obtain intestinal quiet, all cathartics and laxatives are withheld, no food is given, and sufficient opium must be administered to control peristalsis. In our clinic morphin in one-eighth grain doses is given hypodermatically at four hour intervals. This has seemed to control the pain and intestinal activity, without producing any ill effects. Small and repeated doses given at regular intervals of three or four hours have a better effect on the course of the disease and the morale of the patient than a larger quantity given irregularly or only when the woman is in severe pain.

On general principles, all food should be withheld as long as opiates are being given. The vomiting in pelvic peritonitis is of less frequent occurrence and less persistent than in peritoneal reactions due to invasion of the upper abdominal cavity.

The nausea and vomiting usually cease when food and fluids are withheld and *neither nourishment nor medication is given by mouth.* Should the vomiting persist after withholding food and fluids, gastric lavage will generally effect its cessation. Little gagging will be occasioned if a small sized, smooth tipped, well lubricated stomach tube is passed through the nose. This should be connected by a glass T, connecting tube with a reservoir containing a gallon of warm saline or bicarbonate of soda solution. A piece of rubber tubing should be attached to the base of the T as an outflow tube. After the tube is in the stomach, the outflow tube is clamped, and about a pint of fluid allowed to run into the stomach; when a clamp is placed on the tube coming from the reservoir, and the one on the outflow tube is removed, the stomach contents will be withdrawn. This procedure is repeated until the stomach washings are clear and free from bile staining. Performing lavage in this way gives less distress to the patient and assures complete

emptying of the stomach content. *Lavage* also *relieves the gas distention in the upper abdomen.*

Tympany is seldom a very troublesome symptom in pelvic peritonitis, yet gas distention does occur and may cause the patient considerable distress. In most cases the distention may be readily controlled by the routine use of the "Harris drip" after the lower bowel has been emptied of its fecal masses by a small low enema of soap suds.

The Harris drip consists of a gallon douche can reservoir, half filled with a warm solution of five per cent glucose in bicarbonate of soda, or with warm tap water which is kept warm by placing an incandescent lamp in the can. The can is placed on a small stand at the side of the bed, and *the bottom of the can should be at the level of the symphysis pubis.* About two feet of rubber tubing is attached to the outlet of the can, and to this is attached, by a glass connecting tube, an ordinary rectal tube of small caliber. The rectal tube, well vaselined and with all of the air expelled from it, is now introduced into the rectum for a distance of four to six inches. This allows the fluid to run into the rectum and sigmoid until it has reached the level of the fluid in the can, which insures that a single column of fluid at low pressure fills the can, tube and sigmoid.

The level of the fluid in the can changes slightly with inspiration and expiration under the gradual distention of the sigmoid and the obliteration of the sigmoid angle; and, once the sigmoid angle is obliterated, gas usually passes freely back into the can and the contained fluid becomes stained with fecal matter. Gradually the abdominal distention and tension get less and less.

Since we have discontinued the use of large and irritating enemata in our peritonitis cases, distention, pain and thirst have been seldom noted, unless there was an extension of the process; furthermore, the amount of fluid taken up in this way by the sigmoid and colon is sometimes enormous. This naturally increases the blood pressure, stimulates the kidney function and dilutes the toxins, while the absorption of glucose maintains the body heat.

In addition to the foregoing palliative methods, it has been the rule to use either heat or cold to relieve the local pain. *Heat* properly applied to the surface of the abdominal wall *tends to relax the muscular spasm,* increase the local hyperemia, and relieve the pain due to the local peritoneal reaction. Heat also seems to act favorably upon the distention. We have used heat in both the dry and moist form and feel that, when there is any considerable amount of abdominal distention or tension, dry heat acts most kindly.

In using dry heat we employ the Gellhorn baker, first covering the abdomen with a sheet, or, what is better, with a bath towel, and then placing the baker over the abdomen, maintaining a constant heat of 130° to 140° F. This degree of heat can be comfortably borne for hours without burning or blistering the skin. A local hyperemia is produced in the tissues of the abdominal wall, and the stasis favors phagocytosis. It is sometimes amazing to see the relaxation and comfort which come from the continuance of this measure.

Where a Gellhorn or Bier heater is not available, moist heat may be substituted in the form of the hot antiphlogistine or clay poultice. The hot clay is applied directly to the abdominal wall and covered with a layer of cotton batting or six or eight thicknesses of gauze; this, in turn, is covered with a hot water bag which has been but partially filled to lighten the weight. This is held in place on the abdomen and over the poultice by a many tailed binder. These clay poultices need not be renewed oftener than once in twenty-four hours. *Moist heat is particularly grateful when applied over painful exudates.*

Cold is sometimes a better anesthetic than heat when used to relieve the pain over a local inflammatory process. It does not, however, give the degree of relaxation. The too long continued use of either heat or cold can produce serious pathological changes in the skin.

To summarize, the palliative treatment of pelvic peritonitis is rest, starvation, opium and enteroclysis, with the applicaton of heat or cold to relieve the local pain. If this treatment, properly carried out, is doing good, and nature is competent to meet and overcome the advance of the invader, the temperature should fall, the evening elevations should be less each day, the tension, distention and tenderness should diminish, and the leukocyte count and the polymorphonuclear percentage decrease; with the improvement in these general symptoms the general condition and well being of the patient should show marked improvement. *When the patient shows no local or general improvement, palliative and symptomatic measures must give place to surgery.*

A spreading peritonitis is evidenced by the persistence or the recurrence of sharp colicky pains, diffused over the abdomen and not relieved by ordinary doses of morphin; the tension and distention seem intractable, vomiting may persist or recur, the temperature remains elevated, the pulse, due to poisoning of the cardiac muscle, loses its tone and becomes more rapid, the leukocyte count is usually 30,000 or more, and the polymorphonuclear cells over 85 per cent.

Spreading peritonitis demands drainage. Get in, drain, and get out quickly, is the accepted principle. It matters not whether the drainage

is made through the *cul de sac* of Douglas or through the abdominal wall.

During the past winter we have saved several lives in cases of spreading postabortal peritonitis, with seropurulent exudate, by making a free *cul de sac* incision through the vaginal vault and isolating the pelvis (after the Pryor method) by placing small rolls of iodoform gauze in the recto-uterine pouch across the pelvis from side to side. The incision temporarily relieves the abdominal tension and allows the escape of the seropurulent exudate. This diminishes the absorption of toxins, while the gauze promptly excites a peritoneal reaction, as is shown in the rise of the leukocyte count and the relative fall in the percentage of polymorphonuclear cells. These cases have promptly become localized, and the abdominal and general symptoms have subsided.

In others, under novocain and gas, we have made a stab wound incision in the abdominal wall just above the pubis and drained through the abdomen. *There is no need of looking about for the origin of the infection or sponging or washing out the exudate from the peritoneum.* Intra-abdominal pressure and posture will do all that is possible to do in aiding nature to wall off and localize the process. After this simple and direct surgery in the form of drainage has been done, the general plan of palliative treatment already described is carried out until the final issue is obtained. Many cases of spreading peritonitis are lost by allowing them to become diffuse before drainage is established. *If drainage is done promptly and without unnecessary trauma, nature has a breathing spell* and is temporarily relieved of excessive toxin absorption, in which time she is often competent to change the character of the process.

In those cases of pelvic peritonitis which result in a pelvic abscess, the physical signs usually make the diagnosis; for, following the initial symptoms of the acute peritoneal reaction, with the pouring out of a plastic exudate, the uterus is pushed forward and upward by a sensitive, exudative mass, filling the posterior and lateral *cul de sac.* The tumor mass gradually enlarges and forces the uterus above the pubis, while the mass bulges downward along the sides of the rectum and points into the rectum or vagina. The enlargement causes pelvic pain and tenesmus.

Such a pelvic mass is best opened through the vagina by a wide incision in the *cul de sac* through the vaginal vault, as described under posterior colpotomy. *The majority of pelvic abscesses are the result of mixed infection, an infection in which the colon bacillus takes a prominent part.*

CHAPTER X.

TUBERCULOUS PERITONITIS

Tubercle bacilli in the peritoneum—Secondary involvement by the blood stream, by contiguity, or by continuity—No well defined symptomatology—Exudative, adhesive, and caseous forms—Clinical diagnosis difficult—Prognosis—Climate—Tuberculin—Operation.

Tuberculous peritonitis is a primary or secondary infection of the peritoneum by the tubercle bacillus.

Many of the cases of the so-called idiopathic peritonitis are probably tubercular in origin, while the cause is the invasion of the peritoneum by the tubercle bacillus. Certain etiological factors contribute to the conditions and circumstances under which the disease develops.

No age is exempt. Cases have been reported as occurring in the first week of life, while others are recorded in patients of over seventy. The disease is most frequent, however, between the twentieth and the forty-ninth year (Osler). The largest proportion of our own cases have occurred in women under thirty.

From a review of the available statistics, it would seem that about three times as many males are affected as females; but as the gynecologist sees only women, he gets an erroneous impression of the sex incidence. It is generally agreed that, *in the majority of cases in the female, the genital organs are the point of origin,* for Heintze claims that the pelvic congestions of puberty, menstruation, pregnancy, and pelvic peritonitis all predispose to tuberculous peritonitis. Kelly believes that there is a definite relation between pregnancy and tuberculous peritonitis. In twenty-eight per cent of his cases the disease dated from childbirth. In support of this view, Boulland states that the frequent congestion consequent upon menstruation and pregnancy favors the development of tuberculosis, and we all know that pregnancy increases the activity of the tubercular process in the lungs.

Heredity has apparently nothing to do with the occurrence of tuberculosis of the peritoneum, for a family history of tuberculosis is obtained in less than one sixth of the cases. Notwithstanding this clinical fact, Hane claimed to have found a positive tubercular history in thirty-five per cent of his cases. Our own experience has been that tuberculous

peritonitis may attack those who have no tuberculous history and who have been previously in good health.

The disease may be primary, or secondary. Primary peritoneal tuberculosis is relatively infrequent, for a primary lesion can be proved only in the rarest instances. The *fallopian tubes are apparently the most frequent primary seat of tuberculosis in the female peritoneum.* Mayo has reported twenty-six cases. Osler estimates that thirty to forty per cent are primary in the tube, and Konlich places the incidence as high as seventy-one per cent. Even in these reported instances, the possible preëxistence of a primary focus elsewhere cannot be denied.

The secondary form is an extension from other organs. Owing to the rarity of the primary form, it is naturally inferred that when we find peritoneal tuberculosis, it is probably secondary to a tuberculous lesion elsewhere. Therefore, the problem which confronts us is to find the focus and to determine how the infection travels from the primary focus to the peritoneum.

The incidence of tuberculous peritonitis and the occurrence of tuberculosis in other parts of the body, as shown by autopsy records, will throw some light on this subject. Cummins, quoted by Hertzler, in 3405 autopsies collected from the Pennsylvania, Philadelphia, and University hospitals, found some form of tuberculosis in 835, or twenty-four per cent of the cases observed; in addition, 76 showed healed foci. Tuberculous peritonitis was found in 92 autopsies, or 2.7 per cent of the entire number, or in eleven per cent of the tuberculous cases. Nothnagel, Munstermann, and others, confirm these figures; hence it may be assumed that the peritoneum is involved in approximately ten per cent of the cases when death is caused by tuberculosis of some other organ of the body. Kroenig found that the relative frequency of the organs involved was as follows: the lungs in 92 per cent; the intestines in 74 per cent; the kidney in 35 per cent; and generalized involvement in 10 per cent. Cummins' records showed the lungs to be involved in 84 per cent, and the intestines in 32 per cent; the tubes and adnexa in 40 per cent, and the urinogenital organs in 8 per cent. Klebs believes that the intestinal tract is the common avenue of infection. The chief argument in favor of this view is the relative frequency of mesenteric glandular tuberculosis in children. He assumed that the bacilli could gain access to the lymph or blood stream and then gain a foothold in any part of the body, escaping the lymph glands and involving the peritoneum primarily.

When the primary focus is known, the secondary involvement of

the peritoneum may take place by one of the following routes, namely, by the blood stream, by contiguity, by continuity.

An instance of hematogenous infection is where the peritoneum becomes involved as a part of a general miliary tuberculosis.

Extension by contiguity may be illustrated by the approach of the infection from the lungs to the pleura. Here the lesion may be traced along the lymph vessels from the lungs to the pleura. The extension in the lymphatics may take place directly from one lesion to another, propelled by retrograde metastasis, due to the central occlusion of the lymph vessels by the central tuberculous lesion. Contiguous lesions may coalesce and a continuous lesion result.

Extension by continuity is by direct extension without the intervention of any uninfected tissue. The most common example of extension by continuity is infection of the peritoneum by extension through the walls of the fallopian tubes. After the tubercle bacilli have gained access to the peritoneal surface, the dissemination takes place by diffusion and by gravity.

The tubercle when located in the peritoneum does not differ from the tubercle situated in any other tissue. The tubercle is the unit lesion of tuberculosis and may be described as "a small grayish granule, semitransparent, sometimes transparent and colorless, and of consistency a little less than that of cartilage; their size varies from that of a millet seed to that of a hemp seed; in form they are oblong at first glance, but are less regular when examined with a magnifying glass, when they sometimes appear to be angular; they are intimately attached to the underlying tissue and cannot be separated from it without causing shreds of tissue to follow." (Baillie.)

The cause of their oblong form is that their long axis is parallel with the lymph vessel supplying the area affected. This is explained by the fact that bacteria travel most readily along natural channels. The larger nodules result from a limitation of such extension by a process of beginning fibrosis; therefore the larger nodules represent an older process. The very fine lesion is seen in the more acute cases, while the larger tubercle is usual in the case of slowly developing disease. The *tendency of all tuberculous lesions* is to *undergo caseation,* when they lose their transparency and become opaque and cheesy.

In hyperacute lesions the general appearance of the peritoneum may be one of acute hyperemia with edema. The fine tubercles can only be made out on close inspection, but the nodulation may be recognized by the sense of touch. The tubercle bacillus may produce diffuse infiltration without forming circumscribed lesions; thus the intestine may

be thickened to a centimeter or more for a considerable area, without the appearance of tubercles.

In the early stages, when there is a diffuse dissemination of tubercles, the irritation produces an exudation of fluid and ascites is the result. Whether this remains as a simple fluid accumulation, or results in a fibrinous exudate, depends on the amount of fibrin elements which the exudate contains. If these are slight, either from too limited irritation or from the presence of too great toxicity, the fibrin cannot form, and the adhesive type does not result. Excessive fibrin must be deposited to produce the adhesive type. The *adhesive type is the least virulent* of all.

Combination of the serous, fibrinous and caseous types, which are in reality but different stages of the tubercular lesion, are well shown in primary pelvic tuberculosis, in which the tube itself is caseous with fibrinous adhesions about it, and an abundant crop of newer tubercles develop over the remainder of the peritoneum, giving rise to a serous exudate.

The simplest practical classification is that the disease occurs in the exudative, the adhesive, and the caseous form.

In the early stages of the lesion, three characteristics are recognized:

(1) Superficial location,
(2) Small size,
(3) Absence of reaction in the surrounding peritoneum.

Tuberculosis heals by the same process in the peritoneum as in other locations. Bumm has described the healing of the tuberculous lesion by the following steps:

1. Cell infiltration of the tubercle and its environment;
2. Degeneration of the giant cells and epithelioid elements of the tubercles;
3. Increase of the surrounding connective tissue and incapsulation of the tubercles and finally the formation of a scar nodule.

Symptoms.—The *onset is variable and insidious* for there *is no well defined and clear cut symptomatology.* Many peritoneal lesions simulate tubercular peritonitis. The prodromal symptoms may extend over many years, or the onset may be sudden, as in the case of a perforation. *There is commonly a history of general ill health with exacerbations.*

The acute cases begin either as an acute infectious disease, or as an abdominal crisis. Chill, fever, headache, vertigo and malaise characterize

the former type, while vomiting, distention and pain mark the onset of the latter. The conditions may be combined. When fever and malaise predominate, typhoid fever may be simulated. This resemblance is heightened by distention and general abdominal tenderness and pain. In this acute form the chief symptom is continuous high temperature. Sometimes there is a roseolar exanthem and swelling of the spleen. Not all cases of this type begin so abruptly; for instance, a case with rapidly developing malaise may present no other symptom than an increase of girth, or continual bladder irritation may be the first symptom; or, the peritoneal infection may be overshadowed by the severity of the general infection. Complicating pleural and pulmonary involvement are recognized by pain, signs of an exudate, and respiratory disturbance.

The chronic type is characterized by an indefinite onset; the malaise, general weakness, indefinite pain in the abdomen, and possibly intestinal disturbances, may exist for months before the actual diagnosis is made. Local symptoms, as leukorrhea or referred pains, common to irritation of the pelvic organs, dysmenorrhea, sacral pains, may be present when the pelvic peritoneum is involved. Periodic or persistent constipation with localized pain in some region, notably in the region of the umbilicus, less often in the region of the ileocecal valve, may be the first symptoms of the disease. In most cases where there is a sudden onset, the disease is in the miliary stage.

In the fibrinous type, when fluid is present in the peritoneal cavity, the increase in girth may be the first sign; for, notwithstanding the progressive weakness, the patient seems to be gaining flesh. The outline of the abdomen is usually characteristic and the enlargement is general, involving all regions of the abdomen alike. Fluid is first detected in the flanks.

In the purely serous cases, where there are no adhesions, the fluid changes its level with a change of position. Hertzler claims that in tuberculous peritonitis the wave obtained by tapping is less distinct than in other exudates of like magnitude, and that when the position of the patient is changed, the line of the upper layer of the fluid changes less promptly than in other forms of ascites. There may be bulging in the *cul de sac* of Douglas on vaginal palpation. The amount of fluid may not be great, or may be as much as six gallons.

The adhesive type may be associated with or follow the ascitic, or may occur independently. The onset presents much the same sequence of symptoms. Coils of intestines become adherent to each other or to the omentum, which allow pockets to form. These become filled with fluid and present the physical characteristics of cysts. These cysts are

more apt to form in the upper abdomen. When the adhesive type involves the pelvis, a thickened tube may be found to have anchored the uterus, and the bulging fluid, walled off above by the adherent sigmoid, may present a sacculated, semifluid resistance on either side of the tubal ridge. Constipation and digestive disturbances are common.

In the caseous type there is no free fluid. There are bosselated masses, including loops of intestine; consequently there is great disturbance of intestinal mobility. The process frequently ulcerates through the intestinal wall, and a mixed infection follows in the retroperitoneal spaces. The uterus is nearly always fixed, and the omentum, which is thickened and rolled upon itself, presents itself as a palpable tumor. The infiltrated surfaces of the parenchymatous organs, like the liver and spleen, serve to complicate the picture.

Digestive disturbances are at their height in this type. Pain, tympany, and diarrhea occur, while the fever increases as the disease progresses. The umbilicus may be distended, everted, and reddened, showing distended capillaries near its border and radiating veins over the contiguous skin. This sign is of great diagnostic value when present.

Tuberculosis of the pelvic peritoneum is usually associated with a tuberculous endosalpingitis and a perisalpingitis. Primary miliary tuberculosis of the tubes is said to be uncommon. Williams is of the opinion that the disease primarily involves the mucosa, approaching the peritoneum secondarily. This is in accord with the fact that the commonest form is the caseous. Many recorded cases fail to confirm the infrequency of the miliary type. Breaking down of tissue is apt to be early, and once the lesion approaches the peritoneal surface, early attachment to the surrounding surfaces takes place. This explains the formation of the pelvic mass by matting together of the adnexa and adjacent organs.

Clinically, the surgeon must make the differential diagnosis from the small cyst like nodules which form as a result of a chronic irritative process. These cysts are identical in structure with the stalked hydatids so commonly observed hanging from the fimbriated ends of the tubes. They are miliary cysts filled with a clear fluid. The cyst walls are composed of a very fine layer of connective tissue and are lined with flat endothelium and covered by the same kind of cells. They owe their origin to the deposition upon the surface of the peritoneum of any flocculent precipitate and the formation over this of a pseudoperitoneum which gives rise to actual peritoneal cysts. Over the fundus of the uterus these cysts may be as large as peas, or even larger. The larger ones readily collapse when punctured, but the small ones cannot be recog-

nized. The lack of any reactive process about them, either vascular or indurative, and the semitransparent appearance, are sufficient to differentiate them from tubercles. Sometimes there are small granular nodules which appear much like the cysts, but which are made up of granulation tissues covered by a new endothelium layer. These are less transparent than the cysts, and may attain the size of a pin head or a split pea. Occasionally foreign body giant cells are found in them.

Subperitoneal tuberculosis of so slight a degree may exist that its nature may not be suspected until the suspected tissue is sectioned. If it be a tube, it is often possible to detect the diseased area by palpation when it is not discernible to inspection.

The indurative type, in which the subperitoneal tissue is extensively hyperplastic, may closely resemble gonorrheal salpingitis. If caseated areas are discovered, or if tubercles are seen about the border of the process, the tuberculous nature of the disease may be recognized, or section of the tissue will show fine granulations suggestive of tuberculosis. Miliary areas of necrosis are sometimes noted in gonorrheal tubes. A localized thickening of the tube may be caused by tuberculosis, presenting a veritable salpingitis nodosa. These nodules, when sectioned by the knife, present caseated areas. While, many times, tubal tuberculosis and tubercular peritonitis are recognized at the operating table, frequently cases must be followed to the laboratory before a positive diagnosis can be made.

The clinical diagnosis of tubercular salpingitis and peritonitis is difficult. A certain small proportion, *about one fourth, may be diagnosticated by carefully considering the history and physical signs;* but the great majority are not recognized until the abdomen is opened.

In known cases of tuberculous peritonitis, the tubercle bacillus can be demonstrated in only about 50 per cent. The most certain method of demonstrating the bacilli is by injecting the exudative fluid into the peritoneal cavities of guinea pigs or rabbits. Yet this necessarily fails because the bacilli are usually in the tuberculous tissue, and not in the free fluid.

The reaction from the inoculation with tuberculin is unreliable, because the patient may have healed lesions and there is commonly an existing process in some other part of the body. The reaction may be regarded as suggestive and somewhat confirmatory, but not diagnostic.

Very little of positive diagnostic value can be obtained by chemical reactions. Tuberculous exudates may have a higher specific gravity than is found in other ascitic accumulations; on the other hand, the exudative changes in the peritoneum may be sufficient to block some of

the mesenteric veins and thus reduce the specific gravity; hence, reliance on the specific gravity of the exudate is confusing, and has no clinical significance.

The albumin content is greater in tuberculous peritonitis than in other abdominal dropsies; the average is from three to five per cent.

The cell content of the peritoneal exudate in tuberculosis is apt to be richer in small mononuclear leukocytes than in the ascites of cirrhosis or carcinoma.

Judd, of the Mayo clinic, suggests a valuable clinical test to make the diagnosis at the operating table. He pours peroxid of hydrogen into the peritoneal cavity, washing it away with saline solution. The peroxid produces a frosted appearance of the surface, which when removed by the saline, leaves the unaffected portion of the peritoneum a normal pink color, and the tubercles stand out as a pearly white on a pink background.

Theoretically, the leukocyte count should be low, and should aid materially in differentiating tuberculosis from acute suppurating diseases; but experience shows it to be of little clinical value.

Some elevation of temperature is always present in tuberculosis in the acute and subacute stages, if we take the rectal temperature. It is usually highest in the evening, and normal or subnormal in the morning. It may run a course which may clinically simulate typhoid; but the absence of a positive Widal and of the typical rose colored spots excludes the latter. The history of the presence of other tuberculous foci may furnish the clew to the diagnosis.

A large collection of fluid in the abdomen suggests tuberculous peritonitis in children. The subcutaneous veins situated about the umbilicus are distended in 4.4 per cent of the cases.

Pelvic examination shows a diffuse infiltration of the *cul de sac,* or the cervix may be enlarged, soft and cyanotic. I have mistaken tuberculous pelvic disease for pregnancy.

Röntgen examination of the abdomen may be practiced, following oxygen inflation of the peritoneal cavity; involved glands and adhesions are often brought to view by this method, as well as fluid inclusions. It may be of help. My experience is too limited to give a definite opinion, but it certainly appears worthy of further investigation.

Prognosis.—From a review of the literature, it would seem that more persons recover now than formerly; this may be due to the fact that a large per cent are diagnosticated, or because a greater number are operated upon. Certainly the operative treatment has made possible a closer study of the lesions in the early stages. The diagnosis is never

certain without a direct inspection. As it has become generally understood that the operative treatment is the correct procedure, doubtful cases are subjected to celiotomy and thus given the benefit of operation. Hygienic and sanatorium treatment of the primary lung involvement saves many patients from peritoneal complications.

From 65 to 85 per cent recover after operative treatment. Pic observed sixty-four cases of young girls with tubercular peritonitis. Untreated, 50 per cent died. The best results seem to be in the exudative form. The prognosis in the ulcerative and suppurative forms is very grave. The best prognosis is seen in women in whom the disease began in the adnexa and who were treated by removal of the adnexa. Such cases give 75 per cent recoveries in the exudative form and 50 per cent in the adhesive variety. In the poorly nourished, and those living under unfavorable hygienic conditions, recovery is naturally less likely than in those whose constitutional and environmental conditions are more favorable.

Treatment.—The chief agents to be directed against the disease are the natural defenses of the body. Occasionally definite conditions arise where active operative interference is warranted. Where it is possible to remove the focus of infection, operation offers a prospect of relieving the patient of this part of her burden.

The conservative treatment consists in the exhibition of drugs, climatic treatment, radiotherapy, tuberculin, and abdominal paracentesis. The less said about the use of drugs the better, for aside from general tonics, no drug has seemed to have any beneficial or specific effect on the process.

Climate.—Both Leroux and Lalesque are of the opinion that sea air is particularly useful after the acute symptoms have subsided.

X-ray Therapy.—The X-ray has been used more or less extensively with varying results. Rays from hard or medium hard tubes are used, and the patient is given daily séances for three or four weeks; but the available reports fail to show any marked influence of the X-rays on the course of the disease. Direct sun rays, as suggested by Oppenheimer, seem to have beneficial results. He believes that the sun's rays produce a hyperemia of the peritoneum and thus increases the reaction of the tissues to the infection.

Tuberculin.—Little that is favorable can be attributed to the employment of tuberculin injections; our own experience has not led to enthusiasm.

Paracentesis, followed by the use of oxygen gas in the exudative form, has recently been used with claimed success. In cases where the

fluid content of the abdomen can be definitely demonstrated, simple puncture and drawing off of the accumulated fluid, followed by the injection of oxygen gas through the cannula, may produce a slight inflammatory reaction, which may check the progress of the disease. McGlinn opens the abdomen and inflates it repeatedly with oxygen. This is really safer than paracentesis, as it avoids the possibility of puncture of the intestine and injury to the blood vessels.

Operation.—Laparotomy for tuberculous peritonitis is not without danger. The primary mortality has been recorded as between three and ten per cent from collapse, sepsis, and fecal fistulae; besides these dangers, it subjects the patient to the possibility of mixed infection. The exudate is at first beneficial, in that it contains a tubercle antitoxin, which is alone sufficient to effect a cure in mild cases; hence, operation is not indicated early in the process, but after it has become subacute, in the third or fourth month after the onset of the process.

General opinion is in favor of operation after the acute symptoms have subsided. The abdomen should be opened by a free median incision, so that ample opportunity is given for thorough inspection. The fluid is evacuated, but adhesions are better left undisturbed. The application of peroxid of hydrogen or of the tincture of iodin to the diseased peritoneal surfaces has apparently given the best results. Mayo and others insist on the removal of the tubes. By this method he claims twenty-five recoveries; in seven of these, simple laparotomy had already been performed. We believe that in primary isolated lesions of the tube, where local thickening with caseation is present, the tube should be removed; but we cannot see, when the process is more or less general, why the infected tube should be removed any more than we should remove the infected omentum or intestine. We have removed the tubes in tuberculosis of the peritoneum, supposed to be of pelvic origin, when we have failed to find tuberculosis in the tube on subsequent bacteriological examination. Operation probably does good by the active and passive hyperemia which is produced.

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